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## OBSERVATIONS ON CALCIUM DEFICIENCY AS A CAUSE OF CERTAIN DISEASES OF ANIMALS AND MAN.

By C. E. CORLETTE, M.D., Ch.M., D.P.H.,
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AN experiment of fundamental importance, on the calcium metabolism of a goat, was recorded in 1913 by Steenbock and Hart.[1] I need not go here into minute detail, but the experiment was one carried on for a long period, in which the calcium metabolism, with other details, was carefully observed. At the beginning of the period the animal was in milk; the latter part was post-lactational. Important information was acquired as to the calcium balance under the conditions of experiment. The diet varied from time to time for the purpose of estimating the metabolic balance under different conditions, but it consisted of dry food for the whole time except for a period of two weeks near the end of the experiment. The mineral supply of the ingested food was considered more than sufficient for average normal needs, but notwithstanding this, the calcium balance was found to be consistently negative, not only during lactation, but also after the cessation of the milk flow. During this time, the animal was necessarily in confinement. Then it was let out for two weeks on good summer pasture, and exposed, therefore, also to sunlight. No one at that time knew of the peculiar effect on calcium metabolism of sunlight, nor had the concept of a fat-soluble vitamin been matured. But after the two weeks on pasture in the open, the animal was again placed in the metabolism cage, and put on a dietary under which in the previous period a negative calcium balance had been observed. It was now found that the negative balance had become transformed into a positive balance. There have been numerous experiments more or less similar since that time, and all have tended in the same way.

Why was it so? There had been for months a steady drain on the body calcium through the channel of the intestine. There had been very small utilization of the lime—if absorbed, it had been excreted again more abundantly than it had been absorbed—and it was evident that the calcium store in the bones had been steadily drawn upon.

The condition, therefore, was that of a calcium diabetes. No other term expresses it so well, and it can be regarded with advantage from that point of view.

Then, after two weeks on good pasture in the open, this tendency to calcium diabetes was found to have disappeared.

We know now that vitamin *D*, one of the group of three fat-soluble vitamins, possesses the power of stopping this kind of calcium diabetes, and that it is present in good grass pasture. We know also that certain of the ultra-violet rays present in sunlight are able to do the same by activating a lipoid substance present in the animal. The rays actually cause the vitamin to be formed.

If the demineralization of the bones is sufficiently great, it can be recognized by X ray evidence, and later by clinical evidence. It is usually referred to as osteoporosis. In its more advanced clinical forms, when bending and breaking occur, it constitutes osteomalacia. Osteoporosis is found in infancy as an accompaniment of the clinical condition that goes by the name of rickets.

A few words may be devoted here to the concept of rickets. It has become of late years the custom to restrict the term to cases in which one of the conditions is a peculiar abnormality in the X ray appearance at the growth zone. This, for instance, was used as a distinguishing mark in E. Mellanby's[2] studies on puppies fed on various rickets-producing diets. According to Mellanby's use of the term, the change conventionally significant of rickets was only noticed in actively growing animals, and not in those whose growth was slow. In both classes there were signs of demineralization of the bone. But in the latter class, it was no longer "rickets," but "osteoporosis" Now, for the purposes of study it is necessary to conventionalize distinctions. But the mind is fallible, and we are all of us too apt to assume implicitly that a difference of names betokens, at once and throughout, a difference of things. And through looking intensely at a part we may lose sight of the panorama. Neither of the names denoted a normal bone condition, but some degree of osteoporosis existed in either case. The definition of rickets is not a definition of the whole disease from which the puppies suffered.

There is not at present a rigid conventional description limiting the application of the term "osteomalacia." Osteoporosis is a feature common to both osteomalacia and conventional rickets; so is osteoid tissue. In cattle, clinical rickets appears to be specially frequent in the younger animals of a herd whose adult members are suffering from osteomalacia and its accompaniments, such as stiffness, twitchings, lameness, and pica.

### Soil Conditions in Relation to Deficiency Disease in Animals.

In working through veterinary literature on osteomalacia, rickets, and associated conditions of disease, I found my attention over and over again attracted to the character of the soil, climate, and vegetation, in their relation to the prevalence of cases. There seem at first to be utterly incompatible dissimilarities in the conditions that are thought to favour the prevalence of these diseases. According to Friedberger and Fröhner[3] osteomalacia is more common in stall-fed animals than in animals at pasture, and more in certain stables or on certain farms than on others. But it is quite well known as tending to affect animals at pasture under certain conditions, and these conditions may seem dissimilar. The syndrome occurs with special frequency on poor soils, often on sandy or granitic land, or in other cases on hard cement-like clayey soil. Another soil-type is flat, ill-drained, more or less swampy or water-logged land. The disease is

liable to occur on what are described as peaty soils, and, in general, on soils whose characteristic vegetation is coarse and rank. All the soil types enumerated are usually low in their content of calcium and phosphoric acid, and this fact has frequently been alleged as sufficient to account for the prevalence of the disease in them. But that fails to explain why animals pastured on the same land may at one season do well, and at another time show osteomalacia and rickets, with the various other signs of ill-being that precede and accompany these manifestations. Moreover, under certain conditions, signs of the disease can occur on apparently rich volcanic soil. We meet also a curious paradox, on the one hand a tendency of the disease, or disease complex, to prevail in seasons of drought, and on the other hand a prevalence in seasons of constant wet and flooding of the ground.

If we search beneath superficial appearances, we are able, I think, to trace certain interesting features in common. The soil conditions quoted arouse instant questioning. Here are the conditions in which excessive soil acidity is commonly found. And they all agree in being conditions in which soil-nitrification is relatively low and defective. There are two sources of supply through which nitrates for plant food are acquired. One is through breaking down of organic matter by bacterial action through ammonium compounds to nitrites and thence to nitrates; and the other is through fixation of atmospheric nitrogen, dispersed in the soil, by certain special types of bacteria. The earlier stages of the former process appear to be independent of aeration, but the stage of nitration requires oxygen. The nitrogen-fixing bacteria are independent of organic nitrogen, but require aeration of the medium in which they work, and it is to be remarked that an aerobic bacterial flora is a characteristic general feature of soils in which nitration is being efficiently carried on. The soil bacteria concerned with this process require a suitable physico-chemical environment for their working conditions, and the further removed they are from suitable conditions, the less their activity. For instance, they require a suitable temperature; at low temperatures their activity is greatly reduced, and it ceases entirely a few degrees above freezing point. Furthermore, a high acidity interferes with nitrification, though nitrates are said to occur in some highly acid soils. Again, the soil moisture has in various ways a large influence, nitrification being interfered with both by wetness and by undue dryness, and, moreover, the moisture content of the soil must be held in the right way, that is, as a thin film round the particles. A soil of fine texture will be much better than a coarse sand; it holds water without waterlogging, since it offers an enormously greater surface for the film. A cement-like mass of hard clay admits neither of aeration nor of drainage. The physical condition of the soil is therefore in certain ways as important as its chemical constitution.

Soil acidity may be of organic or of mineral origin. In the one, there is an accumulation of acid products resulting from the presence of incompletely decomposed organic matter, as in the peaty type of soil; in the other, there is a mineral acidity derived from the silicates. This would be the acidity found in poor sandy and granitic soils.

Analyses of grasses grown on many poor soils have shown an ash decidedly low in calcium and in phosphorus, or presenting a calcium: phosphorus ratio different from the ideal requirement, so as to make the deficient element of the pair a limiting factor for the other. These grasses have also shown a high silicon content, tending to shift the acid: base balance to the acid side.

C. W. Stoddart published in 1908[4] a suggestive paper on phosphate availability in relation to soil acidity. I may quote here a paragraph:

> Turning now to the phosphoric acid soluble in N/5 nitric acid, it is seen that considerably more is extracted from the non-acid soils than from the acid soils. In other words, the non-acid soils contain more calcium phosphate than do the acid soils. This is to be expected if soil acidity is the cause of the lack of available phosphates.

He further summarizes his results as showing that acid soils lack available phosphate, and states also that figures on humus and phosphoric anhydride in humus suggest that humic phosphorus may be unavailable to plants.

The animal diseases that we have been considering, though common on poor soils, occur also on volcanic soils in which there is abundance of calcium and phosphorus, but it would seem that these soils are then in a highly acid condition. Even soils derived from, and situated on limestone may be strongly acid. And it should be noted that the soil condition may vary at different seasons of the year, so that at one time there is evidence of good nitrification, with growth of good sweet grass, and at another time the vegetation may be poor and withered, or poor in another way, being what graziers would call "sour."

We may refer here to the interesting work of Bottomley[5] on the increase of nitrogen fixation by *Azobacter chroococcum*, a nitrating organism, and on the improvement of plant growth by the action of aerobic bacteria grown in peat which had been sterilized and afterwards inoculated with aerobic forms. This he attributed to water-soluble stimulating substances which he designated "auximones" produced by these aerobes.

Russell and Hutchinson[6] have shown that in some soils there is an excessive profusion of protozoal organisms which exert a very deleterious effect by destroying or inhibiting the growth of bacteria which are necessary to the well-being of the higher forms of plant life.

May I here draw a bow at a venture? It is quite conceivable that beyond calcium and phosphorus there may be some other dietary factor present in good sweet pasture grass, and deficient in pasture grown on acid soils. One can, for instance, imagine the possibility that the sterol which forms and carries the fat-soluble vitamin now known as vitamin *D* has to be derived by animals from plants, and that plants grown on poor acid soils do not

provide it. Perhaps the higher plants have to procure it from soil bacteria that grow with difficulty or not at all in some soil conditions, and well in other conditions. This is mere conjecture, but such conjectures provide points of view, and sometimes form a fruitful basis for study.

However this may be, it is certain that soil conditions have some connexion with food values. Analyses of soil from farms in the Bega district of New South Wales,[7] where many cattle had been suffering from osteomalacia, showed that they were all strongly acid. In non-affected farms the soil reaction varied from neutral to faintly acid. Analyses of the prevailing grasses showed on the affected areas a low percentage of phosphoric acid and lime, and even in the non-affected it was low in comparison with some published analyses of European and American grasses.

The difference to the animal intake would depend on quantity of grass available, on appetite, and on assimilative efficiency. The second and third of these can depend, amongst other things, on vitamin supply, vitamin *B* for the appetite. Probably assimilative efficiency can be affected by all varieties of avitaminosis, but certainly by lack of vitamin *D*. An animal would have to eat more of, and digest better, a food supply low in necessary mineral matter. The supply must always be above the lowest limiting factor, and if assimilation is bad, then the supply must be higher still. When the total calcium of the diet is not far above the threshold point, or critical minimum, it becomes relatively easy to upset equilibrium, either by a decrease in absorption or by an increase in excretion.

In the young growing animal this would be accentuated, as it would also in the breeding or lactating cow, whereas the adult bullock would be able to balance on a smaller intake. A working bullock, if efficient for work, must eat a large total quantity of food to provide calories for expenditure in work. This would bring up with it the gross mineral intake, and if a sterol or potential vitamin were required, the intake would presumably increase in the same way. Thus, if either were dangerously scanty in the fodder, the larger gross quantity of food taken would protect the animal.

### Osteoporosis of Horses: Bran Disease.

In 1905, D. Hutcheon,[8] then Chief Veterinary Surgeon in Cape Colony, contributed an article on "Osteoporosis," illustrated with some good coloured plates, to the *Agricultural Journal of the Cape of Good Hope*, the name being applied to a certain pathological syndrome observed in horses. He quoted extensively from an account by Captain A. H. Lane, A.V.D., on serious outbreaks involving army horses at Wynberg in 1898, and mules at Middleburg in 1904. The diet of the horses was maize seven pounds, colonial oat hay ten pounds, salt half an ounce, bran one pound. It was while on this diet that the animals became affected. It was then changed to oats five to six pounds, lucerne hay eight pounds, green forage four to six pounds, bran two pounds, salt half an ounce, bone meal one and a half ounces. Rapid improvement ensued, and with the exception of some dozen very bad cases out of about four hundred horses, all became workable.

Note the difference in diets. In the former, no green food and a low mineral content, especially low in calcium, which would probably become to a considerable extent a limiting factor for the phosphorus, even if the phosphorus were otherwise sufficient. In the latter diet there was both green forage and an abundant and also a better balanced, mineral supply. In subsequent correspondence, Lane expressed the opinion that the disease was due to a deficiency of phosphates in the food, aided by cold and wet. The mules at Middleburg were cured by a very similar dietary provision. Lane stated that samples of colonial oat hay which had been fed to the mules were analysed and showed a considerable deficiency both of phosphate and of lime, as compared with Transvaal oat hay and average British hay. He stated further, that he was convinced that this disease, usually called osteoporosis in horses, was identical with the osteomalacia of cattle which was so common in South Africa, and which was generally believed to be due to want of calcium phosphate in the food. This opinion is shared by most veterinary authorities, as reference to standard textbooks shows, but it has been disputed by H. B. Elliott,[9] who has described the disease in plantation horses in Hawaii, it was strongly disputed by Hutcheon in the article cited, and it has also been denied by Sir Arnold Theiler, the present Director of Veterinary Research in the Union of South Africa. These three have all expressed their belief that it is due to an infectious process. Theiler says it is so in South Africa, whatever may be the case elsewhere. We may note that similar views were once held about infection in rickets, but the infection theory of rickets is now dead.

Hutcheon denied that diet had any influence on the development of osteoporosis in horses, since it appeared "where the food, care, and management were of the best"—an argument which begs the question. Yet in another passage he remarked in connexion with an outbreak in a racing stable, that the only apparent influences in operation were high feeding and hard training, and that the animals recovered if they were sent away to the country "and fed largely on green nourishing food."

It is a remarkable thing that osteoporosis is also a disease of city horses, when it is sometimes known as "bran disease." The osteoporosis of city horses has the peculiarity that it disappears when they are sent out to grass and reappears when they return to their old stables in the cities. The city horses suffer considerably from founder, or "laminitis" (inflammation of the bed of the hoof), and this also disappears when they are turned out to grass, and reappears when they return to the city stables. The relatively low calcium content of bran, as com-

pared with its phosphorus content, is now so well known as to need no further emphasis. Phosphorus is relatively high in bran, but if its physiological usefulness, or at any rate, its suitability for storage in bone, is limited by a deficient calcium factor, the tissues could be starved of phosphorus in the midst of plenty.

I wish to note one more piece of information from Hutcheon's article, namely, that the appetite of horses suffering from osteoporosis is generally good, and is sometimes described as "voracious."

### The Stijfziekte and Lamziekte, or Osteomalacia and Paralysis, of Cattle in South Africa.

In the year previous to the publication of his article on osteoporosis of horses, Hutcheon contributed an article[10] to the same journal, entitled "Stijfziekte and Lamziekte, or Osteomalacia and Paralysis." He said it affected cattle principally, but also sheep and goats, "and even horses." It was favoured by drought conditions. *Stijfziekte* ("stiff-sickness") and *lamziekte* ("lame-sickness") were regarded as closely related in their origin. The following facts applied to both.

1. Cattle which graze on veld where these diseases prevail manifest an intense craving for bones and all kinds of animal matter. This craving increases as the diseases become more common, and almost ceases when they disappeared.

So intense is this craving at times that cattle have been observed to kill young lambs and eat them, while every vestige of the carcases of those that die are speedily eaten up by the survivors.

2. The animals most subject are heifers with first calves, then cows heavy in calf or giving a full supply of milk, and young growing cattle of both sexes. Full-grown oxen are not so liable, and working oxen still more rarely.

In *stijfziekte* there is great pain on walking; the animal adopts a gait like that a foundered horse, attempting to avoid placing weight on the fore-feet; there is enlargement of the joints, especially from the fetlocks to the hoofs, and the outer digit of the hoofs is larger than the inner one. Elevated circular rings are observed round the hoofs, and in some there is an enlargement above the coronet similar to ringbone in a horse. The animal lies down a great deal, and can scarcely be induced to walk in search of food; then signs of starvation appear, and it generally dies of exhaustion, unless successfully treated. *Post mortem,* there are no indications of disease of the internal organs, but in the articular extremities of the bones, especially towards the feet, there is much congestion. In cases of longer standing the pathological appearances closely resemble those of ringbone and laminitis in the horse. Fractures frequently occur in the bones of the pelvis and thigh. In acute cases, if the bones of the fore-legs are sawn longitudinally, the cancellous tissue at the joint ends will be found dark red and filled with gelatinous blood-coloured exudation. The bone-marrow presents a similar appearance. The bone tissue may be readily cut with a knife.

Hutcheon describes *lamziekte* as a form of paralysis occurring in two forms, a chronic and an acute. In the former, the paralysis is usually confined to the hind limbs, the animal is capable of raising up its fore-quarters, and will drag itself around in search of food. The appetite is unimpaired. It dies usually from exhaustion, exposure, and neglect. *Post mortem* appearances of the bones are very similar to those of *stijfziekte*. The internal organs are usually normal in appearance, with the exception that the contents of the third stomach are much drier than natural.

Let me interpose here the remark that the third stomach is the *omasum,* or *psalterium.* It is the one with leaf-like arrangement, whereby it has gained also the name of the "bible" (Greek *βιβλίον,* a book). The disease here described by Hutcheon as *stijfziekte* and *lamziekte* is acknowledged by experienced veterinary surgeons, for example, R. J. Needham[11] and H. R. Seddon,[12] to be the same as that known as "dry bible" in South Australia, as "impaction paralysis" in Victoria, and as "Midland disease" in Tasmania. Something very like it has been described under the name of "Grand Traverse disease" in Michigan, United States of America, by C. D. Smith.[13] The losses from the disease in parts of Australia have been described as "appalling." They are certainly appalling in South Africa.

Returning to Hutcheon's description, he says that in the more acute cases the animal, when first observed amiss, manifests some nervous excitement, with an unsteady and irregular movement of the limbs. If hurriedly driven while in that condition, it will often drop down suddenly and be unable to rise. In very acute cases the animal is completely prostrate from the start; it may lie with muzzle on the ground, dribbling saliva, in a semi-unconscious condition, lapsing into coma, followed by death.

E Hedinger, Professor of Pathology at the University of Basel, was commissioned by the South African Government about the year 1912 to visit South Africa for an investigation of *lamziekte.* He reported[14] on a detailed study of fifty-two cases. Macroscopically there was hyperæmia of most organs, especially of the intestines; several cases showed circumscribed hyperæmia in the grey substance of the spinal cord resembling the *post mortem* signs in tetanus. In the intestines, especially the small intestine, a mucous enteritis accompanied the hyperæmia. In most cases the third stomach (*omasum*) was dry. Microscopically, (1) the muscles showed extensive fatty degeneration, brown pigmentation, and more or less Zenker's degeneration. The intramuscular connective tissue was often œdematous, sometimes infiltrated with leucocytes and red corpuscles, with circumscribed proliferation of the nuclei of the sarcolemma. (2) In most cases alterations were found in the nervous system. Hyperæmia was often seen in the brain and spinal cord. In several cases an early, but very circumscribed degeneration of several fibres of

the spinal cord was seen. The principal changes were in the peripheral nerves. In most cases these consisted in enlargement of the lymph spaces between nerve and perineurium, then in œdema of the cells lining these spaces, and further in swelling of the axis cylinders and medullary sheath, and lastly, in a formation of myelinic drops. In several cases a very distinct interstitial neuritis could be noticed. (3) In all cases sarcosporidia or Miescher's tubes were found in the muscular system. It has since been shown by P. R. Viljoen[15] that these are found in nearly all South African cattle over the age of one year. Hedinger had suggested the possibility that the sarcosporidia might have something to do with the disease. A similar suggestion as regards a disease of sheep known in Scotland as "scrapie" was made by T. P. McGowan in 1914.[16] In scrapie the sheep exhibit "trotting" movements, incoordination, lameness, paralysis, and itching, and the muscles contain sarcosporidia. I refer to scrapie again later.

Sir Arnold Theiler, Director of Veterinary Research, Union of South Africa, has written a great deal on *stijfziekte* and *lamziekte*.[17] On the theoretical side, he has advanced a number of opinions, but to the present critic, the actual evidence brought forward in support often seems dubious and unconvincing. On the descriptive side, his account of the clinical and pathological appearances corresponds closely with that given by Hutcheon, with a few minor alterations and additions. As regards *stijfziekte,* he has described cases in which the bones did not exhibit any serious amount of decalcification. In young animals growth is stunted, they remain small, have a rough coat, and develope a big abdomen. He follows Hutcheon in confining the term *lamziekte* to the paralytic type of disease. Of this there are various degrees of severity; in very acute cases an animal may be well one day and dead by next morning. He does not confirm Hutcheon's *post mortem* findings on the bones. He describes swelling and redness of the mucosa of the fourth stomach (*abomasum*), some catarrhal enteritis, especially of the small intestine, and sometimes pronounced hæmorrhagic extravasations in the small intestine. In the clinical description it is remarked that in many cases there is bending back of the head and neck, with the head resting on one side; if attempts are made to stretch the head out, it snaps back like a spring. The back may be stretched out straight or bent back, and the animal may be motionless or show spasmodic cramps. In other cases there may be paralysis of mastication and swallowing; the animal desires food and may take it, but be unable to masticate it, and be unable to swallow its saliva. Sensation usually persists; it is only rarely that the sensitiveness of the hindquarters disappears, and usually the animal reacts to pinching or pricking the legs with a needle. It can move the legs, but cannot support its own weight.

Let me again interpose some remarks. It has always been a surprise to me that no one seems to have recognized in these animals—and in many other animals suffering from diseases of various names—that the spastic symptoms described are no other than tetany, and the subtetanic condition which in children is known as spasmophilia. If the symptoms be regarded from that point of view, and in the light of our more recent knowledge of the relationship of the blood calcium to spasmophilia and tetany, a flood of light falls on the whole field of nutritional disease in animals. As we shall see, the way to interpretation is opened up in a marvellous way, although I do not claim that all difficulties disappear at once.

I return now to Theiler's reports. He made extensive inquiries amongst farmers, from whom the following opinions and experiences were gathered. Bone-meal feeding is a useful preventive, but opinion was not unanimous. Green feed is beneficial. The disease tends to be confined to certain areas or certain farms. *Stijfziekte* and *lamziekte* are never found on "sweet veld," but exclusively on "sour veld." There is a good deal of it in a region of limestone and dolomite formation. Bone-chewing is always common where *lamziekte* is common. When bone-chewing is not present, there is no *lamziekte.* A change from sour to sweet veld will cure the disease. Cattle brought from healthy areas into *lamziekte* areas do not contract the disease for some time, ranging from a few months to a year. Working bullocks are least susceptible. Heifers in calf and cows with their first calf are most susceptible. Cows in milk generally go down with *lamziekte* eight to fourteen days after calving. Ostriches are affected by a disease in which paralysis of the legs is the striking symptom.

*Laboratory Experiments on Lamziekte.*

In the Third and Fourth Reports of the Director of Veterinary Research, Union of South Africa, appeared a contribution by Sir Arnold Theiler, H. H. Green, and P. R. Viljoen,[18] describing researches intended to discover whether *lamziekte* was a deficiency disease, evidently meaning an avitaminosis of the nature of beri beri. It is remarked in the introduction that "the superficially analogous disease of *stijfziekte* is not taken into consideration in the paper" since there was no reason to believe that the two diseases, or any of the forms of *stijfziekte,* were of similar origin. The experimental work was begun in 1913 and terminated in 1914, and it should be remembered that the concept of a fat-soluble vitamin, or group of vitamins, had not then been developed. "Vitamin" meant the water-soluble substance we now recognize as vitamin *B,* and whose deficiency induced symptoms of polyneuritis in man, birds, and quadrupeds. The occurrence of paralysis in *lamziekte* had caused suspicion of deficiency of the so-called "antineuritic" vitamin, and the experiments were intended to test the question. Some of these experiments demand special attention.

One series consisted of rice feeding of six cattle, a young bull 2108, grown ox 1928, young heifer 2569, grown calf 2758, cow in calf 3024, cow in calf

approaching term 3039. With the rice was given salt and a small quantity of roughage, at first equal to one pound per day of poor veld hay, and later, autoclaved straw, usually about two pounds per day, but cow 3024 in the later months received four to six pounds per day. We may remark that the diet was certainly inadequate in more than vitamin *B*, being deficient in protein, in calcium, in phosphorus, in the antiscorbutic, and in all three fat-soluble vitamins. The experiment lasted about a year for the animals that survived. In none did any symptom of paralysis occur, and the conclusion was drawn that the requirement for the vitamin must be very small in cattle, and that perhaps the bacterial flora of their own intestinal tract might provide for the necessity, such as it was. As far as vitamin *B* is concerned, the experiment does seem to show that it is difficult to produce the characteristic polyneuritis of *B* deficiency in these animals. The same may be said about scurvy. But the animals showed other symptoms. Gastro-intestinal symptoms were very marked, especially diarrhœa. In may be significant that calf 2758 fractured its femur about seven months after the beginning of the experiment. A point of great interest to me in this experiment was the fact that every animal developed laminitis, which is one of the outstanding features in *stijfziekte*. Theiler and his associates do not seem to have looked at it from that point of view, and record their opinion that it was due to "purely digestive disorder." This is in line with existing orthodox veterinary teaching, but the evidence should be reexamined. It may seem presumptuous in me, but I disbelieve the orthodox teaching. The digestive disorder is, like the laminitis, a consequence, and both are secondary to a common prime cause.

*Field Experiments on Lamziekte.*

Still more interesting is a set of field experiments carried out on a *lamziekte* area. Whereas all the test animals used in the experiments described above appear to have been drawn from healthy areas, in these field experiments it was desired to secure cases of *lamziekte*, and therefore the cattle used were drawn chiefly from areas in which *lamziekte* had occurred, and were placed in camps on a farm called Armoedsvlakte, specially selected for its reputation as a very bad *lamziekte* area. The cattle were allowed to graze over *lamziekte* veld, and various foodstuffs were supplied as supplementary rations to see if the natural occurrence of the disease could be thereby prevented. At the same time, control animals without supplementary rations were employed, and to one lot a rice supplement was given to see what the effect might be. To facilitate a full calorie intake, treacle and linseed oil were used. To supply the "antineuritic" vitamin and improve general condition, bran, beans, maize, and potatoes, were severally tried, also a mixture of bran and bean meal with molasses. With the object of testing the value of yeast, a quantity of this material was regularly administered to one lot.

The solution of these disease problems is of such enormous economic importance, apart from scientific values, that I am sure I shall be forgiven for spending a little time over the details. The animals were divided into lots of ten, yarded at night, fed with the supplementary rations in the early morning, and turned out to graze for the rest of the day. The "orypan" mentioned in the treatment was a commercial preparation of rice polishings, proved in preliminary experiments to be very effectual in preventing and curing polyneuritis in pigeons—the polyneuritis of the variety caused by feeding on polished rice. I give here a *résumé* of the outcome of the experiments.

*Treacle*, Lot C, 10 cattle. Daily ration of two and a half pounds per head, smeared upon hay and fed in troughs. Commenced December 18, 1913, discontinued August, 1914.

On April 8, about four months after commencement, cattle 2976 and 2996 were noticed sick. Number 2976 died April 9, and 2996 on April 13, both of *lamziekte*. On May 16, animal 2989 sickened. It was treated with 100 grammes of orypan, but showed no improvement, dying 4 days later of acute *lamziekte*. On May 20, animal 2623, which had replaced 2996, developed an attack. Ten days later, when on the verge of death, it was put out of misery, and the diagnosis was confirmed. On May 30, the quantity of treacle fed was doubled, but no further cases happened to occur up to the date of discontinuance.

*Linseed oil*, Lot D, 10 cattle. The daily ration of half a pound per head was also fed smeared on hay. Commenced January 6, 1914, discontinued August 10, 1914.

On February 7, animal 2343 sickened, and died two days later. On March 8, 2614 developed acute *lamziekte*, dying the following day. On May 30 the quantity of oil was doubled, but no further cases occurred until June 11, when a mild evanescent attack was diagnosed in animal 2353.

*Beans*, Lot E, 10 cattle. Daily ration of two and a half pounds, fed as soaked whole beans at first, later as coarsely ground meal. Commenced December 18, 1913, discontinued August 10, 1914. On February 11, 2840 was diagnosed as suffering from a slight attack of *lamziekte*, but it recovered a little later. On May 30, however, it developed an acute attack and died in two days. Animal 2843 sickened on May 5, but recovered. On May 30, the daily ration was raised to the very liberal allowance of four pounds per head per day, but nevertheless number 2843 developed a second attack on June 28, of which it died on the thirtieth. Incipent affliction was diagnosed in number 2993 on May 18. Three days later, acute *lamziekte* set in with rapid death. Animal 3170 died of *lamziekte* on June 13, fourteen days after the bean meal supplement had been raised from two and a half pounds to four pounds.

*Bran*, Lot G, 10 cattle. Daily ration of two and a half pounds per head, raised to eight pounds on May 28, generally given dry. Commenced December 18, 1913, and discontinued August 10, 1914.

The first case occurred on January 5, on which day heifer 2139 went down, death ensuing the day after. On March 13, animal 2145 showed signs of *lamziekte* and was treated with orypan. It recovered, but the recovery is believed to have been spontaneous. In May, three more cases occurred. Number 3166, which had passed through a slight attack in February, succumbed to an acute attack on the twenty-ninth, and died within twenty-four hours. On the nineteenth, animal 2998 developed acute *lamziekte*, dying on the day following. On the thirtieth, number 2999 was suspected, but it recovered within a few days, leaving the diagnosis doubtful.

*Mixed Ration*, Lot F, 10 cattle. Commenced January 6, 1914, discontinued August 10.

During the first month, the total supplementary ration for all the animals consisted of twenty-five pounds bran,

nine pounds bean meal, six pounds treacle; or four pound of the mixture per head per day. Thereafter the bran was reduced to ten pounds until the end of May, at which time the whole ration was raised to twenty pounds bran, twenty pounds bean meal, and five pounds of treacle. Animal 2974 died of *lamziekte* on February 7. On May 13, two cases occurred, both terminating fatally within two days, number 2352 dying on the fourteenth, and number 2987 on the fifteenth. In the former case, treatment with orypan was carried out, but with no effect. About May 20, animal 3145 sickened, but recovered spontaneously.

*Crushed Maize*, 10 cattle. From June 13 to August 10, 1914. Daily ration of three pounds per head allowed. This test, it will be noticed, was begun late. Within the experimental period of two months, only one case occurred, animal 3192 dying of acute *lamziekte* on July 26, after having eaten one hundred and thirty pounds of maize in the forty-three days of survival.

*Potatoes*, 10 cattle. From March 19 to August 10, 1914. The daily ration of fourteen pounds per head was roughly crushed immediately before feeding. On May 1, animal 2962 was diagnosed as a doubtful case, but the attack passed off. On May 12, animal 2383 succumbed to peracute *lamziekte*. On May 31, animal 2512 developed a slight attack but recovered. On August 5, number 2962 contracted an acute attack and died the following day.

*Yeast*, 10 cattle. The pressed brewers' yeast was diluted with water, boiled for a few minutes, allowed to cool, and administered. The experiment was started on March 29, but owing to a slip in the schedule the daily quantity of yeast given at first was a quarter of an ounce instead of a quarter of a pound. This, however, was rectified after the lapse of three weeks, and five ounces per head were given each alternate day from April 21 to May 25, after which five ounces were given every day. Animals 2488 and 2603 developed acute *lamziekte* on May 1, dying within two days. On May 18, number 2349 was diagnosed as suffering from *lamziekte*, and on May 21, it died. On May 23, animal 2615 died after two days' illness. Number 2338, put in on May 26, to replace 2615, succumbed to peracute *lamziekte* on June 14.

*Rice*,—Five cattle were started on five pounds of rice per head per day on January 6. On May 16, five more cattle were added, and on May 30 the daily ration for all animals was increased to ten pounds of rice per head. The rice was given soaked. Animal 3137 contracted *lamziekte* on March 20, and died on the twenty-sixth. On May 19, number 3141, of the second batch, after only three days on rice, was noticed somewhat stiff in gait, but this rapidly passed off again, and was not diagnosed at *lamziekte*. Animal 2367 died of *lamziekte* on June 24. Cow 3134 calved on May 26, developed a severe attack on June 28, and was killed on July 3. Animal 2965 also died of *lamziekte* on July 3, after a few days' illness. A fifth case (2441), which died two days after being put into experiment, and was replaced by a fresh animal, is excluded from consideration, as in the initial stages of disease at the time of commencing. The remaining six cattle were not affected up to August 10, the date of discontinuance. The digestive troubles so prevalent amongst the laboratory cattle were evidently prevented by the *ad libitum* grazing in the field.

*Controls*, Lot A. From January 22 to August 10, 1914. Fourteen cattle grazing night and day in the same camp made up the controls. Four cases occurred. Number 3148 went down on May 13, dying on the fourteenth, in spite of orypan treatment. Animal 2328 was diagnosed as suffering from *lamziekte* on May 21, and died on the twenty-fourth. Number 3151 developed an attack on June 3, was treated with orypan on the fourth, but died shortly after. Animal 2323 succumbed to acute *lamziekte* on June 13. A fifteenth animal brought up from Kaffraria on May 5, developed *lamziekte* on June 11. The district from which the animal was drawn is also a reputed *lamziekte* area.

*Comment on the Lamziekte Experiments.*

We see that in these Armoedsvlakte field experiments the animals in most groups received a quantity of "antineuritic" (or *B*) vitamin which should have been an ample protection against the beri beri form of polyneuritis. The particular stock of bran used had been previously tested on pigeons, and had been found to possess an extraordinarily high antineuritic value for those birds. The bean meal had been found highly protective. The protective value of yeast is well known. Yet the presence of a really considerable quantity of these materials in the food had no protective influence at all in regard to the development of *lamziekte*, and orypan, a preparation derived from rice polishings, previously tested on pigeons and found curative, was found quite ineffectual when administered as a remedy in cases of *lamziekte*.

Since it has been found that osteoporotic changes and defects of ossification occur in cases of scurvy, and that Hutcheon, at least, has connected osteoporosis with *lamziekte*, it becomes necessary to inquire whether a deficiency of the antiscorbutic vitamin might not be concerned in the pathogenesis of *lamziekte*. We seem to find this question answered in the negative in the experiments just considered, for it appeared that a liberal supply of crushed raw potatoes did not prevent the disease. In the supplementary rations consumed there was certainly a deficiency of calcium phosphate. If the animals obtained their requirements of calcium phosphate, they must have obtained them from the pasturage on the *lamziekte* farm.

We may turn aside here for a little view of the Armoedsvlakte pasturage as presented in a discussion of the cause of bone-eating by Sir Arnold Theiler in a later paper.[19] Theiler regarded bone-eating as entirely distinct from *lamziekte* in its pathogenesis, and as due to a deficiency of phosphorus in the food. He found that animals with a mild craving declined to touch putrid bones, although they exhibited a craving for bones if they were not putrid. But those with the severest craving would devour them eagerly when rotten. Practically every animal on the farm would chew a perfectly bleached sweet bone. He therefore classified them as follows: Those which ate bones even when putrid were "marked cravers"; those which sought for fresh bone only were designated "mild cravers." The remainder, although they would eat sweet bone-meal if offered, were entered up as "non-cravers." All the cattle at Armoedsvlakte were tested once a week in this way, which permitted the plotting of a curve of seasonal variation. The recording started from May, 1919, when 40% of all grazing animals tested showed craving. There was then a gradual increase until August 5 (winter), when the maximum craving was reached, and 80% of all the cattle ate rotten bones. From that date onwards a gradual descent in the curve was noted, until October 27, by which time the craving had again fallen to 40%. During this period the weather had been continuously dry, but as it grew warmer the spring vegetation began to appear, and this was regarded as responsible for the diminution. During the ensuing fortnight rain

fell, the young vegetation became almost luxuriant, and by the middle of November the craving had rapidly fallen to 6% of all the animals tested—had vanished except for the more or less chronic cravers. This minimum craving was maintained through a fortnight of rainless weather, and then, as the young grass wilted, craving rapidly returned, reaching a maximum of 82% within three weeks. For the next two months of drought it oscillated around this figure. The craving curve up to that date, therefore, showed pronounced craving while the cattle were grazing upon the old grass of the preceding rainy season, reduced craving as the young spring vegetation began to shoot, practically no craving after the beneficial effect of the rains upon the young grass, but a recrudescence of craving during the ensuing drought when the young grass wilted and shrivelled up,—a craving which remained high so long as the drought continued. In the middle of January, 1920, the drought broke, the pasture rapidly recovered, and it was naturally thought that the craving would again disappear. The unexpected happened, however, and notwithstanding the ample supply of good green grass, and the absence of wilting, the craving did not fall to the previous 6%, but only to 50%. As the age of the grass advanced towards maturity, the craving curve again rose, and by the end of March once more stood at 80%.

During the period of initial high craving, hay cut from the grazing areas showed the very small proportion of 0·08% phosphoric oxide. During the period of rapid growth after the rains, the young grass showed 0·56% of phosphoric oxide, or seven times as much. During the period of lowest craving, the phosphorus content remained fairly high, and as the craving rose again the percentage of phosphorus in the grass steadily fell. By January 15, it was down to 0·18% phosphoric oxide.

The figures for calcium oxide were not given. I may say here that the two low figures for phosphoric oxide in the Armoedsvlakte grass are both considerably lower than those found in any analysis of the grasses from osteomalacia areas in New South Wales,[7] calculated to dry matter. Bone-chewing was associated with the osteomalacia in these New South Wales areas.

After this long, but necessary, interruption, let us once more return to the Armoedsvlakte field experiments. It will have been observed that the controls also contracted the disease. The cause would therefore seem to be traceable to something outside the supplementary rations, and resident on the farm. There is the possibility that the animals in the various groups had eaten of some poisonous substance able to cause *lamziekte*. As a matter of fact, that is the way Theiler now explains *lamziekte*.[20] According to his most recent theory, the cattle develope pica as a result of phosphorus deficiency in the pasturage, the perverted appetite then causes them to eat carrion, and in the carrion are certain bacteria which manufacture a poison capable of causing a paralysis which is in fact *lamziekte*. Not all putrid carcases can cause this, but the specific bacteria only occur in carcases in certain regions, which mark the regional distribution of *lamziekte*. Theiler is so sure of his theory that he states it as a complete and final settlement of the question. However, in scientific work no conclusion can be regarded as more than provisional,—and I am wholly sceptical. It may be remarked that, *mutatis mutandis*, Theiler's theory is in all essential respects the same as Lombroso's theory of fifty years ago on the causation of pellagra. The hypothetical cause of pellagra, according to that view, was that it is due to a toxic chemical substance derived from maize affected by some mycotic growth. For many years it was heresy not to believe Lombroso's theory. It is now dead.

We may admit Theiler's claim that he has produced paralytic conditions in animals by subcutaneous injection of toxic material obtained by filtering putrefactive saprophytes, and also by oral administration of the organisms. But at the same time we may remain unconvinced as to the identity of that paralysis with *lamziekte* paralysis. If cows are specially liable (as they are said to be) to go down with *lamziekte* eight to fourteen days after calving, they appear to time their consumption of the toxogenic saprophyte in a very extraordinary way to a period about a week after calving. Why?

### *A New Explanation of Lamziekte.*

I believe I can propose a reasonable explanation of *lamziekte*, and of a whole class of related morbid phenomena in animals, and it seems best to state it here in a categorical form, so as to make it more clear.

Hitherto it has been tacitly accepted by the great majority that polyneuritis, or cognate disturbance of the nervous system, when an indication of nutritional deficiency, belongs exclusively to deficiency of vitamin *B*, the so-called "antineuritic" vitamin. But if I am right, that is not so. It can occur as a result of deficiency of vitamin *D*. It can occur through deficiency of calcium and/or phosphorus in the dietary. There is evidence that it can come from other deficiencies, such as that which is responsible for pellagra. But I am not dealing with pellagra here. It may occur from the deficiency which seems to be responsible for at least some cases of pernicious anæmia. But I am not concerned with anæmia just now.

There is evidence suggesting very strongly that in cases where there is deficiency of vitamin *D*, meaning by "deficiency" a failure to reach the physiological optimum, the intestinal absorption of ingested calcium phosphate is rendered relatively incomplete. Some of this evidence we have seen in Hart and Steenbock's balance experiments on the metabolism of a goat,[1] but much more could be cited, did space allow. In some cases there may be combined with defective absorption an increased calcium loss. How far the gastro-intestinal catarrh that is so frequently observed at autopsy may be responsible for defective absorption I cannot say, but it is not likely to be the whole cause,

for catarrh and erosions appear to be common in every description of deficiency disease. An increased desquamation of epithelium would also pretty certainly increase calcium loss, and if diarrhœa were present, the desquamation would be very considerable. During lactation there is a natural physiological drain of calcium and phosphorus depending on the quantity of milk secreted, and therefore much greater in cows bred for a high milk yield. One may add another remark, that owing to catarrhal gastritis of the fourth stomach there may be lessened secretion of hydrochloric acid, and consequently some difficulty in the solution and digestion of calcium phosphate.

Since reference has been made to possible derangements of absorption or excretion in other kinds of dietary deficiency, we may just note here, but without pushing the analogy, or suggesting explanations that might be quite wrong, the well-established fact that an osteoporotic condition of the bones, together with growth changes at the epiphyseal line, occurs in guinea-pigs suffering from scurvy. Furthermore, it has been reported by Shipley, McCollum, and Simmonds[21] that an osteoporotic condition like that of scurvy occurs in the bones of rats suffering from polyneuritis induced by deprivation of vitamin *B*.

Returning now to vitamin *D*, it may reasonably be conjectured that the more severe the deficiency of this vitamin, the more inefficient becomes the intestinal absorption of calcium phosphate. When, notwithstanding some moderate deficiency in the vitamin, the dietary is such that calcium is very abundant in the dietary, its abundance may be great enough to prevent serious nutritional shortage, or to delay the appearance of signs of trouble. If, however, the dietary be very low in calcium, then the supply of mineral material derived from intestinal digestion would fall below the minimal requirement. Again, we may conjecture with much probability that a wrong ratio between calcium and phosphorus might cause a deficiency of both if one of them is too low. This would be through the operation of Liebig's "Law of the Minimum." If both are abundant, it seems that a wrong ratio would not matter unless some digestive incompetency brought one of these elements below the threshold of sufficiency. In more extreme cases of the mineral deficiency, there would be a failure to obtain a sufficient supply even if the assimilative powers were good. Here, I might say, comes in the great protective usefulness of the symptom called "voracity," "polyphagia," or "bulimia," the last being what it is called in human medicine. It occurs in cases of calcium deficiency, but not in deficiency of vitamin *B*, since in cases of deficiency of vitamin *B*, the more food is taken, the quicker and the worse the catastrophe. In deficiency of vitamin *B*, therefore, anorexia is protective. This feature of the symptomatology is one of the ways in which the polyneuritis of calcium deficiency should be distinguishable from that of the beri beri variety, though it is possible that both deficiencies may be combined. Pica is another protective craving about which much could be written; "licking disease" and bone chewing (osteophagia) in cattle are forms of pica. Coprophagia (eating of fæces) is similarly derived from a protective physiological instinct.

Returning from this diversion, it may be pointed out now that when there is a shortage of calcium (or phosphorus) supply from the digestive canal, so that the outgoings cannot be met, the body must draw on the mineral reserve in the bones. The bones, therefore, form what may be described as a bank.

Touching my conjecture that a wrong ratio between calcium and phosphorus might bring into action the law of the minimum, I am quite well aware that the metabolism of calcium and phosphorus within the body is largely independent. But the demand ratio, nevertheless, is broadly parallel to the ratio in which the two elements are stored. This is well shown by the experimental work of E. B. Forbes and his associates,[22] [23] [24] [25] though limitations of space make it impossible to do more than mention it here. But as though providing for contingencies, we may note that calcium is present in the bones both as phosphate and as carbonate (the latter, however, in relatively small quantity), and we may expect that either reserve could be used as occasion may arise.

Picturing the bone matter as a bank, we may regard the building up (osteoblast) and breaking down (osteoclast) cells as members of the staff, the former on the receiving side, the latter on the paying out side. The working of the bank might be adversely influenced by inefficiency of the regulating mechanism. In the present paper it is not possible to discuss at length what that regulating mechanism is, but it is certain that the parathyreoid glands enter into it, and it appears no less certain that vitamin *D* is some part of it.

The calcium bank might fail through exhaustion of all its reserves. This is too unlikely to consider seriously. More likely would be a failure through exhaustion of the more easily available part of the reserve during a run on the bank. Or, again, a run on the bank overwhelming the present capacity of the staff—the osteoclasts—to handle it, might be imagined as a cause of failure. If we can legitimately draw such a picture, we can imagine a sudden run and a failure to meet it as a "surprise effect." We can picture the milk flow in a newly-calved heavy-milking cow as a sudden run on the calcium or calcium phosphate bank.

When the bank fails, there follows a shortage in the currency; there is a failure of the supply required for the needs of the tissue cells of important organs, and the animal is near to, or over, the threshold of a breakdown. In cases of minor deficiency, there might be merely minor symptoms.

The "surprise effect" is not a fanciful idea invented without physiological warrant. Surprise effects occur in all directions when untrained tissues are called upon to meet sudden great demands.

I am, of course, aware of the physico-chemical view of bone change, addition being regarded as a deposition from solution, and subtraction as a washing out process, a going into solution. Such a conception does not necessarily exclude the operation of "surprise effects."

The extreme and absolutely fundamental importance of the rôle of calcium and phosphorus is hardly realized. Calcium and phosphorus are described by medical writers mainly as the "bone-forming materials," and the general idea seems to be that these elements find their chief use in the building up of the bones, and when that is finished they remain static. Unquestionably they find part of their function in the formation of the skeleton. But to leave it at that gives a wholly inadequate notion of the truth. Phosphorus plays some important part in the nervous system. Again, the profound importance of a correct balance between sodium and calcium ions for the preservation of life and function in protoplasm has been demonstrated convincingly, and many times, by Jaques Loeb and others during the past twenty-five years. Or perhaps I should rather say, the importance of preserving the ratio $\frac{Na+K}{Ca+Mg}$ at the correct balance. Physical and physiological equilibrium rests at the point where the ratio of the monovalent to the divalent cations equals 1:100. The influence of variations of balance of antagonistic electrolytes on phase relations governing permeability of living cells has been discussed in a very striking paper by G. H. A. Clowes.[26] Clowes's experiments seem to show calcium as the king-pin of cell nutrition.

And lastly, there is the abundant and convincing evidence of clinical and laboratory studies of nutrition.

As a matter of fact, the most important part of the functions of calcium and of phosphorus is within the complex of biological processes that makes up the life of the body and its constituent cells. The static function in the bones takes second place.

If I am viewing the position aright, it seems that in osteomalacia and in rickets the osteoporosis is protective, and not primarily an atrophic process, and that the less important is sacrificed to maintain the more important. Decalcification or dephosphorization of the important vital tissues would mean rapid death. But decalcification and dephosphorization of the bones mean weakness, not death. There are other conditions of disease and disuse in which osteoporosis occurs, but we are not discussing that here. But in the osteoporotic changes of osteomalacia and rickets, let me emphasize the view that we behold there a protective process. In rickets, the pathological changes at the growth zone discover an attempt to maintain vital function under difficulties, and from that point of view they are not part of the protective process, but of the protected.

In young animals, a slackening of the growth impulse would slacken the intensity of the bone-forming process at all the growing points. This would give opportunity for better functioning of other vital metabolic processes to which calcium and phosphorus are essential. Thus a slackening of growth helps to solve the difficulties created by short supplies. We can also see that in the absence of growth the conventionally typical pathological appearances held to signify rickets could not appear. The adult would then be held to have osteomalacia, or some non-committal paraphrase meaning the same thing would be used. In a rickety child or animal, a slackening of the pace of growth would obviously tend towards recovery, or "healing."

In starvation, rickets is said to improve. During starvation there is a consumption of depôt fat, and if any fat-soluble vitamin (*A*, *D* or *E*) is present therein, it would be liberated for use perhaps quicker than under other circumstances (Mellanby).[2] Also, there is an autolysis and partial combustion of muscle,—it amounts to feeding with muscle proteins. McCollum[27] sees in this an opportunity for liberation of mineral constituents for supply of body needs during shortage. It would, however, liberate far more phosphorus than calcium, and the ratio would be incorrect for bone formation, though there would be some small quantity of calcium available. I would add that in starvation the specific dynamic action of the food would disappear, lowering the general metabolism very considerably, and, therefore, presumably lessening the demand for some of the calcium and phosphorus. I do not, however, think that this would amount to much.

But in starvation there is another circumstance not hitherto taken into account. The formation of fæces ceases. The stimulation of the intestinal canal is reduced. Its local metabolic changes are materially slowed down, and the denudative process involved in desquamation of epithelium and other material is stopped. This would reduce the output of calcium phosphate very materially, and it would also reduce the output of fæcal fat and lipoid. I showed in 1900[28] that a considerable amount of fat and lipoid is excreted into excluded loops of dog's intestine. Giving the mean results, I found the ether-soluble material to constitute 31·2% of the total dry weights. Of this, 23·5% consisted of fats and soaps, and 7·7% of lipoid, recorded as "cholesterin," as was then usual. The mean ash was 14·34%. I found that calcium phosphate was excreted as bundles of crystals within a desquamating cell, the cell outline being still distinctly discernible. It is scarcely possible to doubt that this is a channel of excretion for the fat-soluble vitamins, in the same way as is the milk of lactating animals.

In cases of experimental rickets, administration of the anti-rhachitic fat-soluble vitamin causes an improvement in the growth zone, so that the conventional signs of rickets disappear. Mellanby[2] noticed that though the "rickets" healed, osteoporosis remained, or might even increase. As I interpret these facts, they show that the "bank"

is working efficiently, and issuing currency promptly and equal to requirements, whether supplies are coming in from the alimentary canal or not. It seems to me that this hypothesis gives the most satisfactory explanation for the continued, or even increased, osteoporosis, while the "rickets" heals,—the important biological function of growth is protected by the abstraction of calcium phosphate from the formed bone.

These rarefied bones may bend or break, and that is a common tendency in later childhood Some of these cases seem to be called late rickets; others appear to be called osteomalacia of childhood.

I shall now put forward a few observations chosen from many more that I have in my note-books. These all seem to have some connexion with questions raised in the course of discussion. I have also records of some interesting experimental work carried out with the aid of farming friends, experiments very successful in practical results, but in some respects very difficult to interpret. There will not be space for this.

### Chalmers Watson's Experiments on Fowls.

Chalmers Watson's experiments on fowls[29] were part of a research intended to discover the relation of meat feeding to gout, and belong to a period before it was known that there were many, or any deficiency diseases other than scurvy. They lasted from March, 1900, to July, 1901.

He fed eight fowls on raw lean meat and water, sand and small grit being supplied from time to time. For the first six months, seven of the fowls were kept in the country in a fowl-run by the side of a wall, the floor being natural soil. The eighth was kept in a cage in the laboratory. They took the meat well, and throve for a few weeks, but then showed lameness. Later they took to resting on the ground in a sitting position. The feathers became disarranged and began to come out. About the same time they showed a marked tendency to pick the lime off the walls. At the end of six months, two were returned to the yard in apparent health. Two had died of acute nervous derangement at the fourth and seventh month, respectively. They went off their food, showed symptoms of paralysis and defective coordination, rapidly became unconscious, and died. Number 3 was killed in the fourteenth month on account of a third attack of paralysis. A photograph of the appearance in a paralytic attack is given in Chalmers Watson's reprints. It is remarkably like Chamberlain, Bloombergh, and Kilbourne's[30] photographs of *polyneuritis gallinarum* induced by feeding polished rice. Number 4 steadily gained weight, but at the end of the fourteenth month it developed symptoms very similar to strychnine poisoning,—opisthotonus, spasms *et cetera*,—but not aggravated by handling. *Post mortem*, it showed signs of tuberculosis. Microscopical examination of the spinal cord failed to reveal changes. The bone marrow was unduly dark. Number 5 manifested at the thirteenth month symptoms of acute paralysis, most pronounced on one side. The general nutrition was good. Number 6 showed poor nutrition, and became paretic. In walking, it raised the right leg higher and put it down more sharply (spasticity?). The *post mortem* examination of Number 3 and Number 6 showed striking changes in the bony system. The femurs were curved. The long bones were thickened and irregular on their surface. The marrow was replaced by what seemed to be osteoid tissue. Chemically, the proportion of organic to mineral matter was greatly increased. The bone marrow in Number 4 and Number 5 was dark in colour.

There is other important matter recorded, but there is no room here to discuss it. Some very good figures accompany the text, at least in the reprints. The best place to study Chalmers Watson's papers is in a collection of reprints appended to his book, "Food and Feeding in Health and Disease," Second Edition, 1913, published by Oliver and Boyd, Edinburgh and London.

### *Discussion.*

Flesh is well known to be remarkably low in its calcium content, though relatively high in phosphorus. Lean meat, such as was given to these fowls, would be very deficient in the fat-soluble vitamins. It is true also that muscle is not rich in vitamin *B* when compared with other sources, but it is able to prevent polyneuritis in birds fed on polished rice. Vedder and Clark[31] found ten grammes daily partially protective for fowls. E. A. Cooper[32] found twenty grammes daily adequate for protection in pigeons weighing approximately three hundred and fifty grammes. As against this, Chalmers Watson's fowls consumed over three-quarters of a pound, or three hundred and forty grammes daily. But nevertheless, they became paralysed. Note, too, that general nutrition, from the calorie point of view, seems to have been good, one excepted. This is the direct opposite of what is seen in vitamin *B* deficiency.

As regards the spastic gait developed by Number 6, I would like to refer to Chamberlain, Bloombergh, and Kilbourne's[30] decorticated rice experiments. Out of their many birds, two, Numbers 5 and 7, did not refuse food, lose weight, and die in a short time of polyneuritis, like the rest. These two fowls ate well and preserved their condition. Number 5, at about ninety days, developed a spastic gait like that of Chalmers Watson's bird, and was chloroformed on the one hundred and fourteenth day. Number 7 did not show spasticity, but was found dead after three months. The experimenters remarked that this instance of spasticity stood alone in their experience and in the recorded literature of similar experiments. I suggest that it was an instance of subtetanic spasticity induced by calcium deficiency, and that if the birds had lived longer they would have become paralysed from that cause, independently of any deficiency of "antineuritic" vitamin.

I might say here that the high-stepping spasticity shown by each of these birds, the meat-fed and the decorticated rice-fed, is also the characteristic symptom in the enzootic stringhalt of horses, to be referred to later.

It was a consideration of these observations recorded by Chalmers Watson that first induced in me, about six years ago, a suspicion that paralysis and other derangements of nerve function might be induced by a nutritional deficiency unrelated to lack of the so-called "antineuritic" vitamin. I suspected that it might be an ultimate result of deficiency of calcium and/or phosphorus in the dietary, or of a deficiency of the fat-soluble factor which controls calcium metabolism. I think it likely, however, that other deficiencies can affect the nervous system in a similar way.

In plain words, these lime-eating, and then paralysed, but not emaciated fowls, presented a condition homologous with the bone-chewing, and then paralysed cattle of South Africa. They developed, if one may call it so, an avian *lamziekte*. The farmer's fattest cattle may go down with acute *lamziekte*.

#### Weill and Mouriquand's Experiments on Pigeons.

An extremely interesting paper was published in 1918 by Weill and Mouriquand[33] on what they called "*Syndrome béribérique expérimental chronique*." Whereas avian polyneuritis of the type induced by polished rice comes on between the twentieth and fortieth day, Weill and Mouriquand, experimenting with pigeons, describe as chronic beri beri a paralysis that did not appear, on their dietary, until many months, sometimes over a year, had elapsed. *Post mortem* examination showed serious lesions (osteoporosis) in the bones. They say "these chronic beri beri symptoms exhibit a complete resistance to medication by raw cortical substance," this substance having been found specific by themselves and others in what they call the acute type. They explained the resistance as due to the fact that in the chronic type an actual nerve lesion had developed, whereas in the acute it was functional. To them it was still a beri beri paralysis. I think they guessed wrong. When one examines the dietary on which this "chronic type" was induced, one finds that it consisted of equal parts of uncooked whole rice, sterilized whole barley, and sterilized whole wheat. This is a dietary extremely low in calcium.

I would say that these cases of avian paralysis resistant to treatment by cortical substance were homologous with *lamziekte*, not beri beri. Like these pigeons, the Armoedsvlakte cattle were also resistant to treatment by cortical substance (orypan).

#### E. Mellanby's Researches on Rickets in Puppies.

Mellanby[2] mentions that nervous symptoms were common in puppies eating his deficient, rickets-producing diets. He classifies them into three groups. In the first, the animals cannot run straight, they may run round and round in a circle, or they seem intoxicated, swaying and falling. In the second group there is paralysis, especially of the hind legs. In the third group the nervous disturbances consisted of tetany and convulsions.

Similar symptoms are described in *lamziekte*, and I suggest that the paralysis and other forms of nerve disturbance seen in these puppies was due to the same essential cause, and homologous with *lamziekte*.

#### Paralysis in Monkeys, and Lions in Zoological Gardens.

As long ago as 1884, Bland Sutton[34] [35] [36] described as rickets and osteomalacia a disease occurring in monkeys and lions at the London Zoological Gardens. The chief symptoms during life were paraplegia and other nervous disorders, and *post mortem* examination revealed great softening of the bones, and pathological changes in the spinal cords of the animals. In more recent years, Campbell and Cleland[37] have described as "cage paralysis" what is obviously the same disease in monkeys and lions at the Sydney Zoological Gardens, though they did not describe the condition of the bones. Campbell and Cleland found that a monkey would improve rapidly on a diet of wheat, maize, and green lucerne. I may remark that green lucerne (alfalfa) provides a foodstuff rich in calcium and rich in vitamin *D*.

As to the reason why caged lions should develope rickets or osteomalacia, I would suggest that it is due to the deprivation of sunlight and of vitamin *D*. The animals are fed on the flesh of worn-out city horses, not animals fed on vitamin-rich fresh grass, but on chaff and bran, and whose meat, therefore, even if fat, would be as poor in vitamin *D* as pig fat has been shown to be by Drummond and associates,[38] except when the pigs have been fed on fresh green food. These carnivores, therefore, are placed under exactly the same sort of conditions as Steenbock and Hart's goat,[1] referred to at the beginning of this paper, and they may be expected to develope the same persistently negative calcium balance as was found to exist in the goat so long as it was deprived of sunlight and vitamin *D*. They would suffer from a calcium diabetes.

#### Phenomena Occurring in Connexion with Lactation.

*Milk Fever of Cows; Lactational Dyspepsia.*

Paralysis and other nervous phenomena coming on after parturition have been noted in many domestic animals. It occurs sometimes in swine. But the phenomena are most severe and most common in dairy cows, and among these, most frequently in those strains which are the heaviest milkers. Apparently it occurs occasionally in cows before parturition; an *ante partum* paralysis has been described also in ewes carrying twin lambs by Gilruth.[39] There is more or less variation in the symptoms, but there is, on the whole, a very striking similarity to those of *lamziekte*. In cows, the acuter manifestations have been termed "milk fever" (though there is not necessarily, or even commonly, a rise of temperature), "parturition paresis," "milk collapse," and so on. In this form, the symptoms come on usually within the first week after parturition, but may be delayed to the fourteenth day. We may compare with this the statement that cows in milk generally go down with *lamzeikte* eight to fourteen days after calving. There is a later condition described in text books as "lactational dyspepsia," or "chronic gastro-intestinal catarrh after calving," which is associated with nervous symptoms of a less acute kind, but which by their description seems to be essentially of the same general type, and probably of the same pathological significance, since the same very specialized treatment benefits it.

The onset of milk fever is sudden, with signs of discomfort and obvious illness. The senses are dulled, the walk is unsteady and straddling, and typical of stringhalt, or the legs may sway and cross each other, and they soon give way. The animal comes to the ground, where it lies in a

drowsy state with head resting on shoulder, flank or ground. Occasionally there is excitement or disordered movements, with cramps, and flexion and extension of the limbs. Apparently at first there is not true paralysis, but spasticity. In favourable cases, after from one to four days the animal may get up and resume feeding, the suddenness of the improvement being as marked as the suddenness of the attack. Some, however, are restored to intelligence and appetite while the hind limbs remain paralytic or paretic for days, or even weeks. In other cases there may be signs of paralysis of the soft palate and larynx, and swallowing is impossible, the somnolence increases, the corneal reflex disappears, and the animal dies comatose. According to Law,[40] the death rate in untreated cases is as high as 70%.

In lactational dyspepsia there is dulness, diminished appetite, imperfect rumination, constipation, diminution of the flow of milk, and staring coat. In recently calved animals the body seems permeated with a sickly odour, the milk while warm carrying the odour intensely. Nervous phenomena usually increase, dulness becomes more marked, and the animal staggers if made to move. In other cases the animal, which has for a few days seemed out of sorts, begins to "paddle" with the hind limbs, and walks with a spastic stringhalt gait. Licking of the walls may be noticed. The animal may fall in a convulsive spasm and remain down, suffering from recurring convulsions. Later on the body may remain in a sort of permanent tetanic spasm. In other cases the animal is simply unable to get up. Cerebral excitement often replaces torpidity. Death may be from exhaustion, suffocation, or inanition. It is remarkable that the standard textbooks on veterinary medicine (Hutyra and Marek, Hoare), state that tetany is practically unknown in domestic animals; nevertheless, I am bold enough to assert that what is described in these animals is tetany. It is worthy of very special remark that this lactational or *post partum* dyspepsia is said by Begg[41] to be most frequent in artificially fed cows, and to be practically absent when they are at grass.

In recent times a very successful empirical treatment of milk fever has been introduced. It is known as Ever's modification of Schmidt's treatment, and it consists in passing a cannula up through the orifice of the teat, and pumping air into each one until the udder is tightly distended. It is left in that distended condition, the operation being repeated if required. The almost invariable result of the treatment is that in two or three hours the cow gets on her feet, the bowels move freely, urine is discharged, and water and food are taken (Law[42]). The same treatment is said to be beneficial in lactational dyspepsia (Hoare[43]).

In the absence of any reasonable explanation of the almost miraculous effect produced by this remarkable treatment, I have set about finding one. I believe I have succeeded, but it fails to be a solution if my hypothesis as to the cause of the symptoms is incorrect. Upon making inquiries from men with practical experience in the treatment, I found what I expected to find, that the distension caused a sudden and relatively enormous reduction in the quantity of milk secreted. My conjecture is that milk fever, or milk collapse, is due to a denudation of the readily mozilizable calcium, or calcium and phosphorus, though it is less likely to be the latter, since there is probably more readily mobilizable phosphorus than calcium in the tissues. This denudation is the result of a sudden surge of the calcium towards the milk glands, and is a "surprise" effect. There has been an unusual run on the calcium bank, or calcium phosphate bank, in the bones, and the osteoclasts, the paying-out staff, have been for the time being, unequal to the demand. Hence the breakdown. A temporary reduction in the milk flow gives time for readjustment of supply to demand by reducing the outgo of calcium phosphate.

The so-called lactational dyspepsia which does not occur in cows at grass might be well accounted for by deficiency of vitamin *D* in stabled cows on dry fodder, and especially easily if it is a low-calcium fodder, such as oats grown in an acid soil. This would reproduce in the cow exactly the same conditions as in Steenbock and Hart's goat,[1] already referred to, and it would accordingly induce a persistent negative calcium balance,—calcium diabetes,—whether she were lactating or not.

I suggest that temporary recovery from the symptoms of milk fever and lactational dyspepsia would be obtained by intravenous injection of 200 mils of 2% sterilized solution of calcium chloride, or alternatively, by intramuscular injection of parathyreoid extract prepared by Professor Collip's method. The first would add calcium to the circulation for a few hours, the second would mobilize calcium phosphate from the bones.

The efficiency of the paying-out staff in the calcium bank in the bones is affected in some way by the activity of the parathyreoid glands, and the activity of the parathyreoids seems to depend on the presence of vitamin *D*. At least, that is the way I have come to look at it after some years of close study, but I cannot discuss that further here.

### Epizootic Stringhalt.

I am acquainted with a district in New South Wales where the farm animals have from time to time been liable to show various morbid conditions. For instance, slow growth, lameness, stiffness, incoordination, various rheumatoid affections, gastro-intestinal catarrh with very foul-smelling fæces, coprophagia, geophagia, stiffness and paraplegia in pigs, and, in fowls, the laying of soft-shelled eggs. But I pass over these to refer to one special feature, described as stringhalt.

On one of the properties, the horses have, at times, for many years shown a propensity for eating the dirt of the paddock around their watering place. These horses, like others in the district, exhibited a symptom known as stringhalt. I may add that the horses pastured on this place have been liable to develope stiffness in the legs and lameness for at least thirty-five years past. Also when driven in harness they had to be handled with great care, because on turning they were liable to fall over,

with symptoms resembling vertigo. The soil on this place is of a highly acid type.

The kind of stringhalt here referred to assumes epizootic proportions in certain regions at times. It has been described from Victoria, New South Wales, and New Zealand, and its nature and cause have excited a good deal of speculation. A detailed clinical description of it was written many years ago by the late E. Stanley.[44] Stanley found the disease to occur on cold, wet soils and sour herbage. Reakes[45] has described the disease in New Zealand, and his article is illustrated with two photographs showing the action of the horse. I have seen a description of exactly similar symptoms from Nova Scotia by W. H. Pethick,[46] though it was not called stringhalt.

Now, I suggest that this stringhalt is a subtetanic condition homologous with what is known as spasmophilia in children. Further, it is due to calcium deficiency, and will be found to be associated with a hypocalcæmia.

#### Geophagia, "Sand Disease" of Horses, and Coprophagia.

Geophagia, or earth-eating, is manifested by many animals suffering from nutritional disorder. It is, for example, shown by animals deprived of common salt, and such animals seek out the most brackish soil. It is seen in children suffering from cœliac disease and allied conditions, diseases characterized by the occasional appearance of tetany as a symptom. Some of these children have an instinct sufficiently well-directed to choose mortar or other lime-rich material (Still).[47] A condition known as sand disease occurs in horses in many lands. These animals suffer from gastro-intestinal, nervous, and other symptoms, and sand is found in the intestine after death. Sand disease was a feature exhibited by the osteoporotic horses at Wynberg camp, referred to earlier in this paper. Where these morbid conditions most abound, the prevailing soil will very frequently be sand, and this may account for the kind of soil found in the animals. Grass or fodder grown on sand is fodder grown on a very acid soil, and would be poorly supplied with calcium. Therefore, unless animals can eat very large quantities (polyphagia), they are liable to suffer from calcium deficiency. When feed is scarce, there can be no polyphagia, so they will then be specially liable to show symptoms of deficiency disease.

There is, however, another resource. If the animals cannot get unlimited fodder, they can still get calcium phosphate by coprophagia,—and if it is any use to them, they can get lipoid and the fat-soluble vitamins from the same source,—just as they would from milk. On this view, coprophagia is due to a protective biological instinct. According to my information, coprophagia was widespread among army horses during the war. They were on dry feed—oats and grass hay—and appeared to maintain good health. Some would put that all to the credit of the official rations. Considering the facts, such a conclusion would be illogical. If my view is correct, the coprophagia preserved the animals in the presence of an imperfect ration.

#### Liver Disease.

I may say that there is reason to believe that in calcium deficiency there are degenerative parenchymatous changes in the liver, in line with the state of epithelial tissues everywhere else in the body. Liver disease is constantly associated with nervous symptoms. These would be merged in the general complex of nerve symptoms in deficiency disease. I have referred to liver disease of this causation in an earlier paper,[48] and the subject is too large to pursue further here.

#### Some Cases of "Forage Poisoning" from a New Point of View.

An enzootic of horses and mules was recorded in 1915 from the Griffith farm, near Lexington, Kentucky, by Graham, Himmelberger, and Pontius,[49] which was considered to be forage poisoning. A number of horses died during a period of four months after exhibiting the following symptoms. dulness, partial paralysis of the pharynx, salivation, weakness and unsteady gait, and "various nervous manifestations," followed by prostration in a few hours. An animal once down was unable to rise unassisted. They observed the same symptoms on three other farms. Feeding experiments with the various feeds from these three farms gave negative results, but on the Griffith farm they reproduced the symptoms by feeding with oat hay from the farm. They oats appeared to be clean and good. We are not told what kind of soil the oats grew on, but it should be remarked that oats grows well on poor, peaty soils, that is, highly acid soils. This is the soil to produce oats with the lowest possible calcium content.

Next remark the clinical picture,—one bearing an extraordinary similarity to that of milk fever and of *lamziekte*.

Next let me quote from the authors, whose minds were occupied entirely with the toxic theory, the following:

> Feeding experiments with the various feeds from these farms resulted negatively. In such cases it is probable that only a limited amount of the feed might contain the fatal material, or a marked tolerance to the same might prevail in some animals. The fact that some animals are markedly resistant is illustrated by one of our experimental horses. This animal was a hearty eater, and consumed approximately ten times the quantity of oats consumed by any one of the other experimental animals. In all experimental horses, with this one exception, the feeding of the oats terminated fatally.

Their explanation puts a strain on the probabilities. That the animal which ate ten times the quantity of a presumably toxic material should be the only one to escape, is too great a paradox. The reader should now refer back to my account of the Armoedsvlakte experiments, and he will there find comparable fatalities. To account for the recovery I offer my explanation of the protective value of polyphagia. I conclude that these farm horses died from deficiency of calcium phosphate, not from a food intoxication, and that the farm corresponded

to what would in South Africa be called a *lamziekte* farm.

### Ergotism.

Ergotism is described in veterinary literature as liable to affect animals under certain conditions. In this connexion, ergot is often used as a generic term to include other fungous growths on grasses, believed to be more or less similarly harmful. It is said to cause nervous symptoms (incoordination, spasms, paralysis), sores in the mouth, about the feet or elsewhere, loosening of the hoof, gangrene of ears, tail, or feet, and abortion. I can believe that ergot is poisonous, but there is, to my mind, good reason for being extremely sceptical about the diagnosis of these morbid manifestations as due to ergot or similar fungous parasites. If ergot can produce gangrene and the other symptoms, experimental feeding with ergot has been extremely unsuccessful in proving it without fallacy, or even in getting any results at all. And most animals will not voluntarily eat ergotized rye; they seem to prefer starvation.

Law[50] in his great veterinary textbook says that:

> Towards the end of our long winters in the Northern States we occasionally find widespread gangrenous ergotism from eating infested hay.

But we are surprised to read a little further on that:

> Usually ergot and smut can be safely fed in relatively large amounts with a liberal ration of potatoes, turnips, beets, green fodder or ensilage.

So it takes nearly the whole winter before the ergot takes effect, and the effect can be warded off by adding other materials without taking away the supposedly toxic material. It is much more likely that the vitamin *D* of the hay had lost its activity, and quite possible also that in many of these cases it has been a low-calcium hay. But can lack of "fat-soluble vitamin" produce all these symptoms? The answer is that it can and does do so in laboratory animals, which may exhibit stomatitis, sores about the feet, loss of cuticular structures, gangrene of toes, ears, and tail. If that nutritional weakness were reinforced by severe cold, the effect would be much more prevalent and much more severe. These animals, supposed to be ergotized, were in precisely the same case as Steenbock and Hart's goat.[1]

Gilruth[51] records that cattle, sheep, and horses, pasturing in the Hawke's Bay and some other districts in New Zealand, are very subject to nervous symptoms in late summer and autumn. This was noticed especially, or solely, in pasture mainly composed of seeding rye grass. The animals exhibited muscular supersensibility (spasmophilia ?), and incoordination. It was popularly attributed to ergot, but Gilruth seemed rather doubtful. I would point to the fact that rye-grass is another fodder plant that flourishes in very acid soil, and that under such circumstances it would contain very little calcium. If animals are confined to such pasture, they might suffer from deficiency, like the bone-chewing cattle on the Armoedsvlakte pasture in late summer.

A few words may be said here about ergotism in man. I have a strong suspicion that certain human epidemics that have broken out in times of famine have been homologous with the *lamziekte* of cattle, cage paralysis of monkeys, and so on. Human osteomalacia is a disease of chronic privation, but I do not mean this so much as the "convulsive" type of ergotism (*Kriebelkrankheit*), and lathyrism.

According to Hirsch,[52] epidemics of so-called ergotism have been of two kinds, the gangrenous and the convulsive. These two kinds do not occur together. I have nothing here to say of the gangrenous type, but I am bold enough to hazard the opinion that the convulsive form was tetany, due to a dietary limited to grain (rye), and not much of that, a dietary, in fact, similar to that of Weill and Mouriquand's pigeons. The spasticity of *lamziekte* animals I have already attributed to tetany.

That convulsive ergotism was accompanied, more or less, by itching, as its German name *Kriebelkrankheit*—"itching disease"—shows, causes no difficulty. It makes it rather like the Scottish "scrapie" of sheep, the "trotting disease" of sheep in Germany. But Halsted's[53] experimental studies of partial thyreoidectomy (really partial thyreoparathyreoidectomy) in dogs showed that intense itching and falling of the hair occurred in a number of his cases, together with twitchings and general convulsions. It is now well known that the blood calcium is greatly reduced after parathyreoidectomy, and that complete removal causes rapid death from tetany. Having said this, I suggest now that scrapie and trotting disease are also homologues of *lamziekte*. This disease, or these diseases, prevail on damp, sour pasture.

A word might be said now about the "mange" that Mellanby[2] found so severe and troublesome in some of his puppies receiving deficient diet. Is it not rather significant that Erdheim[54] found "mange" to be very troublesome in his rhachitic rats? Then let us think again of Halsted's experiments. Was the "mange" simply an accidental accompaniment, or was it a manifestation of hypocalcæmia? I suggest that it might have been, but of course it remains possible that it was one of the varieties of parasitic mange. Is it possible that some of the cases of "mange" in domestic animals are manifestations of calcium deficiency?

### Lathyrism.

As to lathyrism, that is also a spastic, convulsive, and paretic condition occurring only in famine times, and attributed to the effect of a dietary confined rather closely to lathyrus peas—and those probably not in great plenty. It is well known that seeds, including peas and beans and all grains, are very low in their calcium content. A man or animal confined mainly to a diet of this kind would get relatively little calcium, and, like the so-called

ergotized people, he would be in the same case as Weill and Mouriquand's pigeons.

But peas and beans of all kinds are relatively deficient in a constituent that is essential to life, namely cystine, the important sulphur-containing amino-acid. This has been shown in a long series of papers by Johns and Finks, and it has been shown by the same experimenters[55] that cystine deficiency is liable to occur in animals on bean diet, unless the beans are boiled, or unless cystine is added. Since for human use peas and beans are boiled, the danger of cystine deficiency is minimized, but it is different with animals.

The most complete description of lathyrism in recent times has been contributed by R. Stockman.[56] Stockman remarks that:

> As very large numbers of mankind habitually eat lathyrus peas, it is certain that when they are taken as part of a mixed dietary no poisonous symptoms occur, but in times of famine or poverty, lathyrism is apt to occur.

Lathyrism is said not to occur in man or animals when the peas are only a subordinate constituent of the dietary. As a symptom of lathyrism, the horse is said to suffer from "roaring," due to paresis of the recurrent laryngeal nerve.

Stockman made a series of experiments on monkeys, rabbits, pigeons, ducks, pigs, and sheep, feeding them for a prolonged period on lathyrus peas. He produced symptoms in the monkeys, but not in the other animals. The peas were boiled or steamed, and though described as given "exclusively," it appeared that in the monkeys there was an addition of fresh raw fruit and "vegetables." As to the symptoms produced, one of the most prominent was tetany, displaying the characteristic "*accoucheur* position" of the hands. One of the monkeys certainly died of tetany. I cannot see evidence of anything in the symptoms except indications of calcium deficiency—which they would have shown on a grain diet quite as much as they did on the peas. Stockman regarded the symptoms as lathyrism. If I am right about calcium deficiency, the symptoms were the same as those of calcium deficiency, and Stockman's conclusion that the experiments proved toxicity in the peas is founded on fallacy.

As regards the effect on animals, I cannot see in paralysis of the recurrent laryngeal nerve any more evidence of toxicity in the peas than of calcium deficiency. Sjollema[57] says rabbits become bald in cystine deficiency, and that the baldness is cured by giving cystine. Hair contains about 2% of cystine, so it is easy to see why deficiency should cause baldness. It is not recorded that horses lose their hair through eating lathyrus peas.

Yet perhaps cystine deficiency could be produced in animals fed on lathyrus peas instead of grain. Hart and Peterson[58] have investigated the sulphur content of various fodders. They noticed that the sulphur of straw did not disappear on ignition, but remained in the ash. But a large part of the sulphur of grain disappeared. This they interpreted as showing that the organic sulphur was practically all in the grain. The logical inference is that the cystine is in the grain, and that straw contains very little. Accordingly, an animal fed on straw and grain would depend on the grain for the necessary cystine. If the grain were replaced by peas, it is possible to see how a cystine deficiency could occur. The risk would be minimized by mixing grain with the peas. Whether this ever occurs is another matter. But what becomes of the toxic theory here?

### A Concluding Warning.

I have mentioned, but perhaps not sufficiently emphasized the point that in calcium deficiency we have a panorama, a general disorder involving all functionating cells, and therefore involving the important organs of secretion, internal and otherwise. When we think of the rickets, gastro-intestinal catarrh, diarrhœa, bronchitis, and the like, we should think of them always as parts of a panorama, not all of which is clearly visible, and as representing a process going on everywhere, including the liver, the pancreas, the kidneys, the thyreoid, the parathyreoids, and so on. The function of these organs, like that of the nerves, is disordered and depressed. The glycogenic function of the liver is disturbed, and its detoxicating function more or less suspended. The animal is brought to some extent into the condition of an animal with an Eck fistula, in which the portal blood is diverted into the systemic circulation without passing through the liver. Metabolic poisons may not be properly detoxicated. It is small wonder that grave symptoms occur.

And I would say one more word. I have reason to believe that these organs, liver, pancreas, kidney, parathyreoids, and also the nervous system, do not always recover completely when the original deficiency is removed. Some permanent damage often remains. Thus a cirrhosis of the liver in animals may be the result of a deficiency no longer existing in the dietary. Cœliac disease in children may be due to the same thing.

### Summary.

Calcium and/or phosphorus deficiency can cause nervous disorder closely resembling that produced by deficiency of vitamin *B*. It is, or may be, implicated in many morbid conditions of man and animals in which nervous symptoms often occur. Amongst these are polyphagia, earth-eating (and "sand disease"), bone-chewing, coprophagia, "ergotism," "lathyrism," scrapie, or trotting disease of sheep, epizootic stringhalt, laminitis, rickets, osteomalacia, spasticity, paraplegia, tetany, milk fever and lactational dyspepsia of cattle and other animals, and the *stijfziekte* and *lamziekte* of South African cattle, and conditions answering to the same elsewhere. To these may be added some cases of supposed forage poisoning.

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# Reviews.

## CONCISE OTOLOGY.

THE handbook by Richard Lake and E. A. Peters on diseases of the ear maintains the high standard that the previous editions held. It is a small, compact volume of 289 pages, well illustrated and printed in clear type.[1]

The brief description of the anatomy and physiology is clearly put, that of the inner ear being especially helpful. The chapters on the general and special examination of the patient are concise and indicate the best mode of procedure to follow in order to gain a thorough understanding of the local lesion.

The different tuning fork tests are well described and the series of the subdivisions of Rinné's test described under the name of the proportional test indicate how a classification of the types of defective hearing can be arrived at. This is original work and definitely helps the surgeon in forming an opinion of the probable lesion present.

Acute and chronic inflammatory affections of the middle ear are described in a clear way and the most efficient mode of treatment is indicated in each case. The intracranial complications are also described in a brief but lucid manner.

In the chapter on the diseases of the internal ear the essentials are incorporated, but it appears rather brief for clear understanding.

The principal operations on the ear are described in a short but clear manner.

In the appendix there are some prescriptions given under the different headings, which will be found very useful. The value of X ray films in the diagnosis of mastoid conditions does not appear to have received the amount of attention to which it is entitled.

Altogether this small manual should be most useful for the student desirous of obtaining a working knowledge of diseases of the ear in a short time. To the general practitioner it will be found most helpful, for it presents the essentials within a short space and in such a manner that they may easily be found.

[1] "Handbook of Diseases of the Ear for the Use of Students and Practitioners," by Richard Lake, F.R.C.S. (England), and E. A. Peters, M.D. (Cantab.), F.R.C.S. (England); Fifth Edition; 1927. London: Baillière, Tindall and Cox. Demy 8vo., pp. 328 with illustrations. Price: 12s. 6d. net.

## SURGERY IN THE TROPICS.

"TROPICAL SURGERY AND SURGICAL PATHOLOGY," by Karuna K. Chatterji, is a comprehensive work on pathology and treatment of the main surgical conditions peculiar to the tropics.[1]

In the case of the two principal tropical surgical complaints—hepatic abscess and filariasis—the pathology has been treated in a most exhaustive manner and the methods of dealing with amœbic abscess leave nothing to be added.

In dealing with filariasis the author does not appear to have devoted sufficient attention to acute retroperitoneal lymphangitis which so often resembles an abdominal crisis. Other filarial conditions are well dealt with, however, and he describes a new method of operating on hydrocœle which in early cases is better than either excision or eversion.

The surgical treatment of acute filarial conditions is usually unsatisfactory or even impossible, but he describes a number of palliative measures which are useful in the relief of symptoms till the attack has subsided.

In dealing with the subacute and chronic conditions, he has very carefully gone into details of the various operative procedures.

The illustrations of filarial conditions are good and instructive to practitioners who are not previously acquainted with the condition.

The portions of most interest to practitioners in the Australian tropics are those dealing with filarial hydrocele, retroperitoneal lymphangitis and chyluria.

The cases of elephantiasis so frequent among native races are very rare in Australia, but moderate elephantiasis of the scrotum and penis are occasionally met with and for the operative treatment of these cases the author has described an excellent procedure.

The descriptions of the tropical granulomata and their treatment, while only of little interest to practitioners on the mainland, are of great help to practitioners in Papua where these diseases are prevalent among the native population; the illustrations in this section are very helpful for diagnostic purposes to those previously unacquainted with these diseases.

The account of the operating room procedure is instructive, but many anæsthetists will disagree with him when he prefers chloroform or alcohol, chloroform and ether mixture to ether. Most tropical surgeons, however, will agree with his remarks on the seasonal variation of septic organisms in the operating theatre and on the lowered resistance of patients and the increased infectivity of the skin during certain months of the year.

## THE LARVÆ OF ANOPHELES.

"THE ANOPHELINE LARVÆ OF INDIA, CEYLON AND MALAYA," by Professor C. Strickland and Dr. K. L. Choudhury, is a very useful publication.[2]

This book has achieved what it set out to do. In the first place the authors demonstrate the fact that the capture of anopheline larvæ is not the easy and simple matter that some people believe. Secondly, there are excellent keys of simple construction, accompanied by drawings by which one may readily identify any given larva of an oriental species of *Anopheles*.

Special attention has been paid to the method to be adopted in the carrying out of an anopheline survey. Details of the methods to be employed in catching the larvæ, bringing the catch home, breeding the mosquitoes and identifying the adults are given and in addition that for the keeping of an efficient and permanent record. The popular belief that an inexperienced person may go out to a

[1] "Tropical Surgery and Surgical Pathology," by Karuna K. Chatterji, F.R.C.S.I., with a Foreword by Major-General Sir R. Havelock Charles, G.C.V.O., K.C.S.I., M.D., LL.D., M.Ch., F.R.C.S.I., I.M.S. (Ret.); 1927. London: John Bale, Sons and Danielsson, Limited. Royal 8vo., pp. 274, with illustrations. Price: 16s. net.

[2] "An Illustrated Key to the Identification of the Anopheline Larvæ of India, Ceylon and Malaya, West of Wallace's Line, with Practical Notes on Their Collection," by C. Strickland, M.A., B.C. (Cantab.), and K. L. Choudhury, M.B., D.P.H. (Cal.), with a Foreword by Sir Ronald Ross, K.C.B., K.C.M.G., F.R.S.; 1927. Calcutta: Thacker, Spink and Company. Imp. 8vo., pp. 67. Price: Rs. 4/8.

swamp with a dipper in one hand and a bottle in the other and perform an efficient anopheline survey is shown to be erroneous. The authors also stress the fact that the worker who neglects to identify the larvæ which die in the breeding jars, commits gross negligence.

The reader will find much that is useful in regard to clothing to be worn when engaged on survey work. "The gentlemen who turn out for field-work in full dress with all medals up, as they would for the King's birthday parade, should be dismissed at once." These are very appropriate words which should be taken to heart by those afraid of getting their feet wet and their clothes soiled.

The book is well got up, lucidly written and full of useful practical information, even to those not domiciled in the Orient.

To those engaged upon malaria surveys this work is indispensable.

## THE CHEST IN THE HEALTH.

"THE NORMAL CHEST," by Meyers, is a book of practical conception.[1] It collates in some four hundred readable pages the fundamental facts about the thorax and its function in the normal individual of different types.

Much of medicine is empirical. An endeavour in this book is made to place knowledge of the normal chest on a scientific basis, to create a clear conception of normal anatomy and to lay the foundation of a clear understanding why specific pathological changes produce specific symptoms.

To the student this book is of particular value, because it collects in an organized way the information scattered through a multitude of textbooks wherein it is not co-ordinated and where much of its practical value is lost.

To the graduate, this volume is an inspiration. It arouses consciousness to the large amount of clinical information, passed unheeded or unrecorded daily in ignorance or haste. This information is of immense value in diagnosis, prognosis and in so far as it presents a clear conception of causation, an aid to treatment.

The book is divided into twelve chapters. The first two chapters give a *résumé* of anatomy of the chest, osseous, muscular, vascular, visceral and the influence of anatomical relations on symptomatology.

Particularly clear and helpful is the description of lymphatic drainage and glandular distribution, the influence on surface anatomy created by physique and the surface distribution of referred pain.

It deals with the mechanism and physiology of respiration. Physical examination is reviewed and the value of various chest signs is discussed. A short review of cardiology is undertaken with special reference to electrocardiography and orthodiagraphy.

Important methods and details of radiology are recounted. Monographs with special reference to pædiatrics, innervation, development and acoustics conclude a volume which is brief and comprehensive and which will well reward perusal and purchase.

## THE PROBLEM OF THE NEUROTIC.

THE author of a readable and instructive work on personality has produced a complementary study on the neurotic.[2] Dr. Gordon who has approached his subject essentially from the dynamic standpoint, emphasizes the need for remembering that neurotics are personalities, not diseases. The Kraepelinian disease-entity concept has served a useful purpose, but no longer represents the latest in psychiatry, so that there is a demand for well balanced expositions of recent advances in the study of neurosis. Hitherto we have had to turn largely to America for literature dealing with the application of the newer psychology to the problems of mental disorders, a province in which the teaching of Adolf Meyer has proved fruitful and inspiring. This book from the pen of an English psychiatrist is specially welcome.

Dr. Gordon has avoided the excesses both of the Feudian school and of the mechanistic psychologists. Life is not all sex or conditioned reflexes. The neurotic is described as characterized by failure of adaptation both within and without the *ego*, as possessing a loosely knit, self-regarding sentiment or, if physiological terminology is preferred, as being poorly integrated. In his review of the Freudian theory which is both concise and impartial, Dr. Gordon ventures to hope that before long the concepts of psychoanalysis will be brought into closer association with other departments of knowledge and that they may even be interpreted physiologically. Freud himself and some of his followers have prophesied that many psychological problems will one day be solved by endocrinology. In dealing with the work of Jung, Dr. Gordon devotes considerable attention to psychological types, a topic already dealt with at greater length in "Personality." Adler and Janet also receive attention. Separate chapters are devoted to the outstanding types of neurasthenia, anxiety states *et cetera*. There is an excellent study of the underlying personality in hysterical and dissociation syndromes. Other chapters deal with "Physician and Patient," "Patient and Public," "Body and Mind." There is much of practical value in the sections on the scope and limitations of psychoanalysis, suggestion and persuasion. This book which is one of the International Library of Psychology, Philosophy and Scientific Method, is not intended solely for the medical profession and psychiatrists may find little that is really new to them. On the other hand the general practitioner will find it well worth his while to look to this book for help in dealing with the not inconsiderable number of neurotics who come to him for help. Psychologists and social workers will also find the volume of value.

## THE CONTROL OF CONCEPTION.

A REVIEW of the first edition of Dr. Stopes's book on contraception was published in THE MEDICAL JOURNAL OF AUSTRALIA of January 31, 1925, at page 113. There have been several reprints since and recently a second edition has appeared, larger than the first by sixty pages.[1]

Dr. Stopes has emphasized the fact that the book is addressed to medical practitioners and from this point of view, as was said before, its subject matter shows evidence of a sincere attempt to point out the indications for contraception and the best methods of its attainment. Many case histories are given; in some instances these seem to show the necessity for careful antenatal supervision rather than for the prevention of conception.

The attitude of Dr. Stopes towards the subject may be gathered from the reference to "the man who has got the crank idea that union should be for procreation only" and other passages may be taken as indicating the views of the advocate and not those of the impartial scientific observer. In addition to the medical aspect of the question there are also social, ethical and national points of view that must be considered very carefully. It seems hardly correct to say in the words of Arnold Bennett, quoted by the authoress, that "the main present obstacle to the complete success of the birth control movement in Britain is not the arguments against it nor the reactionary irrational opposition which confronts even beneficent and simple plans for the amelioration of mankind, it is the notorious false shame of the Anglo-Saxon race."

Still the book is full of information on this contentious subject and may be read by medical practitioners with interest and profit. It is necessary for them to know something of the problems of modern life and the subject of this volume is a very live one and occupies a prominent position in sociological and medical literature.

[1] "The Normal Chest of the Adult and the Child, including Applied Anatomy, Applied Physiology, X-Ray and Physical Findings," by J. A. Meyers, in collaboration with various writers; 1927. Baltimore: The Williams and Wilkins Company. Royal 8vo., pp. 434, with illustrations. Price: $5.00 net.

[2] "The Neurotic Personality," by R. G. Gordon, M.D., D.Sc., F.R.C.P. (Edinburgh); 1927. London: Kegan Paul, Trench, Trubner and Company, Limited. Sydney: Angus and Robertson, Limited. Royal 8vo., pp. 310. Price: 12s. 6d. net.

[1] "Contraception (Birth Control), Its Theory, History and Practice: A Manual for the Medical and Legal Professions," by Marie Carmichael Stopes, with an Introduction by the late Professor Sir William Bayliss, M.A., D.Sc., F.R.S., and Introductory Notes by Sir James Barr, M.D., LL.D., F.R.C.P., Dr. C. Rolleston, Dr. Jane Hawthorne and Obscurus; New and Enlarged Edition; 1927. London: John Bale, Sons and Danielsson, Limited. Royal 8vo., pp. 506. Price: 15s. net.

The Medical Journal of Australia

SATURDAY, FEBRUARY 18, 1928.

# The Reform in Medical Education.

MEDICAL schools acting to some extent under the compelling influence of the General Council of Medical Education and Registration of the United Kingdom adopt a curriculum of medical studies and establish a standard for the several tests. The curriculum is modified from time to time to permit of the inclusion of fresh subjects and new points of view, but the plan on which it is built is retained. The tests or examinations vary as the curriculum is changed. It would seem that those who are responsible for the training of medical students, lose sight of the fundamental objective of the medical schools. Very little is gained by requiring students to spend five or six years learning in parrot fashion the elementary facts in the exact sciences on which the so-called practical application of these sciences to medicine is based. Medical education should comprise the moulding of the mind of the student in such a manner that he may recognize the object of his existence as a practitioner. To achieve this end he will be taught to challenge every doctrine in medicine and to examine its chemical, physical or biological soundness. He will realize that his task is to take a wide view of health and to contribute in the general endeavour to maintain it throughout the community. His interest in disease will gradually recede as the intrinsic and extrinsic factors disturbing health are traced and understood. Fifty years ago the medical practitioner was required by his patients to treat symptoms rather than disease. The pathological conditions capable of cure were few and little attention was paid to the pathogenesis and ætiology of disease. It is true that the experienced practitioner of those days gave excellent service to his patients. He gained an intuitive sense of his patient's condition, even if he believed in many erroneous things. Subsequently the science of bacteriology, the application of biochemistry and biophysics to medical problems and the development of epidemiology gradually changed the attitude of the practitioner toward disease. The disease process could be attacked and in some instances healed; the cause could be eliminated and in this way Nature was given an opportunity of effecting a cure; the defect could be covered and a temporary or even permanent reestablishment of normal physiological conditions could thus be effected. At present the medical profession has reached a stage at which the intrinsic and extrinsic causes of disturbances of physiological processes can be removed not only in the individual, but even in a community. In other words medicine has passed from the phase of the treatment of symptoms, through the phase of treatment of disease to the phase of prevention.

It is obvious that in these circumstances the foundation that served for a curriculum is no longer of value. The whole scheme must be rebuilt. Before the question of the curriculum can be considered, an agreement should be reached in regard to the general aims of medical education today and for the next two or three decades. It is necessary to anticipate events. It must be recognized that it might be necessary to introduce reforms to apply to students only in their first year. This would mean that it would be six years before the full effect of the reforms would be reflected in the medical profession. A conference of accredited representatives of each of the three medical schools could be held with advantage to formulate the general scheme, to suggest an outlined curriculum so as to insure relative uniformity and to examine the difficult question of the tests of knowledge and ability to discharge the functions of a medical practitioner.

If an agreement could be reached in regard to the general objective of medical education, there should be but small diversity of opinion concerning the plan on which the curriculum should be formed. The modern teacher will hold the opinion that the student must continue his studies of chemistry and physics throughout the whole course. The pathology of disease, its causation and the proper method of attacking it must depend entirely on these fundamental sciences. Moreover, no advance would be possible if the student were taught the minimum

amount of biochemistry and biophysics to enable him to understand the present day hypotheses of diseases, such as diabetes, hyperthyreoidism or rhachitis. If he has a grasp of the subjects in not too elementary a form, he will be able to follow new work without the necessity of revising his scientific knowledge and he will also be in a position to contribute to a better understanding of the pathogenesis. In the next place the new curriculum should be drafted with due regard to the possibility of attacking the ætiological factors. The old idea of leaving preventive medicine to the specialist in the health departments of the several governments must give place to the new plan of making this subject the chief part of a general practitioner's work. At the present time it is almost impossible to persuade the general practitioner to take up preventive medicine in earnest. That can be achieved only after the mind of the student has been moulded to this idea. It will depend on the outlook of the leaders of educational thought whether the reform will be thorough or merely a makeshift. Men with wide vision, clear minds and unlimited determination are needed. The prevision of Anderson Stuart and of Harry Allen is essential for this reform. Even when the proposal was made to scrap the curriculum and to build again from new foundations, those in charge revealed that they lacked courage and imagination. Perhaps a conference of representatives will be the means of bringing forward a leader who will infect his colleagues with some of his enthusiasm and driving force. The reform will not be complete until a leader is chosen who demands a scheme according to which the student will be able to think for himself and cease to learn his lessons by rote, often in the same words as are used by his teachers.

The replacing of the ordinary examination by a more logical test will be the last step in the reform. The written and to some extent oral examination is an anomaly at the present day. The amount of intelligence, industry, initiative and ingenuity exercised during the year is of greater importance than the display of the same qualities for an hour or two in the examination room. The real test should be of full understanding, not of verbal memory.

# Current Comment.

## PERTUSSIS.

THE toll of lives claimed by pertussis throughout the Commonwealth of Australia is relatively large. During 1926 the deaths numbered 340. Of these 158 were of males and 182 of females. The number of deaths in each of the several States was as follows: New South Wales 132, Victoria 121, Queensland 61, South Australia 3, Western Australia, 22 and Tasmania one. The death rate was six per 100,000 of population. In pertussis as in other infective diseases, the totals vary considerably from year to year. From 1922 to 1925 inclusive the annual totals for the Commonwealth were 182, 119, 420 and 463. In one State only, South Australia, is pertussis a notifiable disease. The importance of the subject is obvious and no apology need be offered for reference to it in these columns. The clinical aspect has been discussed by several authors. Readers are referred to an excellent account given by Southby in the issue of April 16, 1927. His discussion of the symptoms, diagnosis, complications and treatment leaves little to be desired. While the clinical point of view is most important for the practitioner, the bacteriological and pathological aspects must not be neglected; these serve as the foundation on which the clinical superstructure can be built.

The *Bacillus pertussis* which is generally regarded as the cause of the disease, was described by Bordet and Gengou. It is commonly held that this organism is found in the mucous membrane of the respiratory tract, but chiefly in the remoter parts. It is found more often below the larynx than above it and is more abundant at the beginning of the whooping period than later. It has also been stated that if cultures are taken only from the upper part of the respiratory tract, the organism may be overlooked as it grows very slowly in the first cultures and its growth may be obscured by that of other microorganisms. In some work the account of which was published in July, 1927, Lawson and Mueller, of Harvard Medical School, used a modification of the original Bordet-Gengou medium. They found that the optimum reaction range for growth in the medium was pH 6·6 to pH 7·3 and that this reaction was approximated with sufficient accuracy by adding to the unadjusted Bordet-Gengou medium sufficient blood to make the final concentration 5% or 10%. Lawson and Mueller examined cultures from 1,115 suspected patients, contacts and patients with pertussis. The *Bacillus pertussis* was found to be present in 259 or 23% of the 1,115 cultures. They had records of 533 proved cases of pertussis and in 219 of these the bacillus was recovered. Included in this series were 23 patients who had never whooped and every one of these yielded a culture which contained the causative bacillus. Some useful lessons may be learned when these findings are considered together with a recent contribution by Lawrence W. Smith.[1]

[1] *Archives of Pathology and Laboratory Medicine*, November, 1927.

Smith has studied eight infants, suffering from pertussis, who came to hospital with a complicating pneumonia. Cultures were taken and blood counts were made every day or every other day. These eight patients were included in a series of 3,000 cases occurring at the Boston Floating Hospital. The eight patients died and *post mortem* examinations were carried out. In four cases a definite diffuse broncho-pneumonia was found and cultures in which the *Bacillus pertussis* predominated, were obtained from the lungs. In the other cases the pneumococcus, streptococcus and *Bacillus tuberculosis* were "equally important." In one of the first mentioned four cases definite bronchiectasis and peribronchitis were found. In one instance there was an extensive change of the epithelium from the usual columnar ciliated form to a squamous type of epithelium. In one instance in which diffuse broncho-pneumonia affected both lungs, there was a concomitant hyperplasia of the tracheo-bronchial lymphatic glands with lymphadenitis. The trachea and bronchi were pale and œdematous and a few submucous petechial hæmorrhages were present. Histological examination revealed a diffuse bronchopneumonia with a muco-purulent exudate containing relatively little fibrin. There was considerable desquamation of the alveolar epithelium with an accompanying lymphocytic infiltration of the peribronchial tissues and the alveolar walls. This mononuclear reaction was also found within the alveoli. The most striking lesion in this case was the presence of myriads of minute cocco-bacilli which presented the morphological characteristics of the *Bacillus pertussis*. There was little evidence of any severe toxic necrosis. The lesion was apparently a direct extension from the bronchi which appeared similarly infested with microorganisms. In discussing the pathological changes, Smith points out that the fine differences between a lung affected by pertussis and one involved in some other form of bronchitis are sufficient usually to make a differential diagnosis possible by means of X ray examination. In approximately 80% of the 3,000 patients there was demonstrable peribronchial thickening, involving chiefly the lower branches of the bronchial tree. During the course of the disease there was an appreciable diminution in the peribronchial shadow. Concomitant with the peribronchial thickening there was an enlargement of the tracheo-bronchial lymphatic glands. As a rule this gradually subsided, but complete reversion to normal was often delayed for many weeks or even months. In a few instances permanent enlargement of the glands with fibrosis was seen after as long a period as three years. Smith points out that in arriving at conclusions of this sort it is necessary to have a knowledge of the normal X ray appearances of the chest before the onset of the disease. He has been fortunate enough to be able to do this in approximately three hundred persons and he states that his observations support his general conclusions. Although he is not prepared to be dogmatic on the subject, he gives it as his general impression that the peribronchial shadow became less more rapidly in patients who were treated by exposure to X rays than in those not so treated.

In four of the cases described by Smith cultures were obtained *post mortem* in which the *Bacillus pertussis* "predominated." He does not state what the other organisms were. It is not likely that the broncho-penumonia was the result of the sole activity of the *Bacillus pertussis*. It is safe to conclude that the part played by other bacteria was not inconsiderable. Smith apparently is not prepared to concede this, for he states that "it is apparent that the importance of the pertussis bacillus itself as the principal cause of many of the secondary complications, especially broncho-pneumonia, has not been sufficiently recognized." In one of Smith's eight cases the most pronounced findings were those of acute miliary tuberculosis and he supposes that a hilum tuberculosis became "lit up" as a result of the superimposed pertussis. In other words the resistance of the patient was lowered and the pathological changes in the lung were favourable to the growth of the tubercle bacillus. It is justifiable to conclude that the broncho-pneumonia in the other cases was caused in a similar fashion. It is true that in the acute miliary tuberculosis case no pertussis bacilli were found at autopsy and that "myriads" were found in the other cases. In the latter instances the large numbers of bacilli indicate a poor antigenic response invoked by them; their presence does not exclude the action of some other organism causing the pneumonia. It must be remembered that all the *post mortem* examinations described by Smith, were carried out on the bodies of patients who had died as a result of pneumonia complicating pertussis. It would be interesting and doubtless instructive to have complete examinations made of the lungs of patients, suffering from pertussis, who died from an affection of some other system. It is often difficult to isolate bacilli from the secretions of the respiratory passages. If it were not so, it would be possible to obtain a higher percentage of results than those obtained by Lawson and Mueller.

There thus arises the question of the importance of bacteriological examination in cases of pertussis. Lawson and Mueller have shown that suitable culture methods will reveal the organism in a large number of cases and that bacteriological examination can be used to control the quarantining of patients. They also obtained cultures of the bacillus from patients who had never whooped. It is the whoop which always attracts the attention at any rate of the laity. Bacteriological examination has been used so extensively in the diagnosis of diphtheria that the public accepts and expects swabbings to be taken when that disease is suspected. If medical practitioners can be induced to have bacteriological examinations made in suspected pertussis, the public will fall into line and progress will be made. In Australia but little work of the kind has been done. Both the bacteriologists and the morbid anatomists of the Commonwealth can by accurate and systematic observation and report do something towards the elucidation of the problem and the eradication of the disease.

# Abstracts from Current Medical Literature.

## MEDICINE.

### Diagnosis of Abdominal Disorders in the Aged.

A. MÜLLER DEHAM (*Wiener Medizinische Wochenschrift*, April 9, 1927) considers from his *post mortem* experiences that many diseases of the gastrointestinal tract in elderly persons are overlooked. This is largely due to their lowered reaction. Diseases which are associated with high temperature and increased pulse *et cetera* at younger ages, are more prone to produce merely a slight reaction in the aged. Complaints regarding digestion usually have their origin in some other organ than the stomach. Two groups of gastric carcinoma can be noted. In one the symptoms and physical findings are classical, but the prognosis is better than in middle age owing to the lowered general reaction. In the other the symptoms remain doubtful except for cachexia and occult blood in the stools and the diagnosis is made only *post mortem*. Gastric ulcer is mainly latent and is frequently unaffected by diet. Pain in the gastric area may be due to emphysema or chronic dyspnœa associated with cardiac or bronchial asthma. Cholelithiasis is so frequently found in elderly women as to be considered normal. In cases of jaundice the differential diagnosis between carcinoma and stone may be difficult. All forms of diarrhœa may be observed and it is mistaken for incontinence of the lower bowel. Constipation generally causes no symptom and in many instances is nervous in origin. Rectal carcinoma is often missed. Ileus is not common and is usually due to an incarcerated hernia. While appendicitis is unusual, the classical symptoms are generally lacking and even in the presence of peritonitis there is little pain and no generalized rigidity.

### Diet Rich in Liver in Treatment of Pernicious Anæmia.

G. R. MINOT AND W. P. MURPHY (*Journal of the American Medical Association*, September 3, 1927) report the observations made upon one hundred and five patients suffering from pernicious anæmia who were fed upon a diet rich in liver. In all instances the response to liver feeding was prompt, the patients improving rapidly. The first response to the diet seems to be a temporary and definite increase of the reticulocytes, antedating any increase in the number of the red blood cells. But no matter in what form of relapse the liver diet commences the red cells seem to increase at the same rate and rise to the same height. The increase of corpuscles outstrips the hæmoglobin concentration and the colour index falls; if the latter phenomenon does not occur, the patient usually does badly. Coincidently with the increase of the red cells, the leucocytes show a satisfactory rise, often to well above normal and there is a rapid decrease of the bile pigments of the plasma. It would appear that the benefits derived from the diet are not transitory; the improvement in the numbers of cells has been shown to continue for two or three years. Apart from this the œdema, palpitation, weakness and general irritability rapidly diminish, the yellow tint of the skin vanishes and a pink colour appears. Furthermore, the improvement in the characteristic neural symptoms is frequently gratifying and gastro-intestinal disturbances rapidly disappear. As to the rationale of treatment, it is thought that liver, as well as kidney, stimulates maturation of the megaloblasts which crowd the bone marrow of pernicious anæmia patients during a relapse. It is found that the requisite amount of cooked mammalian liver to be given daily is two hundred grammes.

### Gastric Ulcer.

F. W. WHITE (*The American Journal of the Medical Sciences*, May, 1927) reports the results in the medical treatment of fifty-one patients suffering from ulcer of the stomach and followed up for a period of five years or more. They are taken from his private practice. Accuracy of diagnosis is essential in attempting medical treatment and with the aid of X ray methods it is possible to bring the order of accuracy up to 90% or even higher. The selection of patients for medical treatment depends upon such factors as the youth of the patient, the shortness of the history and the absence of complications such as hæmorrhage, obstruction or perforation. Treatment is by means of frequent feedings six times a day or more with a bland diet. The author insists upon slow and complete mastication. For the first week milk and egg, milk and cream or cereal gruels are allowed. The diet is gradually increased as the symptoms abate, but raw fruit and vegetables are forbidden for six months. Rest is important and the first two weeks are spent in bed. The drug treatment is limited to alkaline powders given in between feeds and these may be continued for as long as twelve months. Atropine is given by the mouth in doses up to one milligramme (one sixtieth of a grain) once or twice a day to check hypersecretion. Bismuth is not used. Tobacco is forbidden. Special attention is paid to possible sources of infection, such as the teeth, the tonsils *et cetera*. When the patient feels well, special care is taken to keep the treatment up in regard to diet and tobacco; therefore an efficient follow-up system is necessary. X ray examinations and tests of the fæces for occult blood are carried out at regular intervals. Should the presence of occult blood be noted in the fæces after the first week of the treatment, surgical treatment is considered. The question of cancer developing in the base of an ulcer is discussed and the author's experience is that such an eventuality is rare, only 2% among his patients. He suggests that the condition in these individuals was malignant from the beginning. All patients whose condition is complicated by obstruction of the pylorus, should be treated surgically, unless there is no residue after six hours. In all cases of gastric ulcer, whatever the treatment, it is necessary to exercise great care in following up the patients for years and determining their condition by means of X ray examinations and by tests for occult blood.

### Cardiac Failure.

A. E. C. KENNEDY AND T. OWEN (*The Quarterly Journal of Medicine*, July, 1927) discuss the limitation of muscular effort and its relation to cardiac failure. Physiologists consider that the limit of muscular effort is determined by the cerebral distress due to anoxæmia. Clinically, modern opinion is that cardiac overstrain is rare in young people and when it does occur, it is in the form of transient heart failure in a heart previously damaged by infection. Excessive exercise does not damage the healthy heart and repeated excessive exercise does not lead to cardiac hypertrophy. Lewis states that in dilatation of the heart, previously attributed to heart strain, the basal condition is an unrecognized paroxysm of tachycardia, generally the result of infection. After Marathon races the heart size has been shown to be reduced rather than increased and collapse of competitors seemed to be of a nervous type, in conformity with the teaching that general fatigue is localized within the nervous system. Several experiments were carried out on healthy young men who were persuaded to do standing running (two hundred or more steps per minute) for some minutes until exhausted. It was found that diminished oxygen (16%) or increased carbon dioxide (5%) in the inspired air, caused in the first case earlier exhaustion and in the second mental confusion and ataxia. From the latter effect it appears that increased carbon dioxide in the blood or lungs is not the cause of exhaustion in athletics or violent exertion of any kind, since the mind remains clear in such cases. The cause appears to be the rise in hydrogen ion concentration, due to accumulation of lactic acids as the mechanism for oxygen intake fails and of carbon dioxide as the mechanism for elimination of carbon dioxide fails. The limitation of voluntary effort was set by the eventual failure of the cardio-respiratory mechanism to prevent a rise of hydrogen ion concentration to a point at which voluntary effort ceased. In the men tested there was no sign of congestive heart failure when exhaustion occurred; they ceased to be capable of further exertion, but not on account of heart failure. When the heart is diseased on the other hand, excessive exertion is undertaken, the heart cannot maintain

the circulation in the same way as in health and eventually fails to supply sufficient blood to the lungs for the carrying on of satisfactory respiratory exchanges. The increased work at the same time induces forced respiration and the two together lead to blood collecting in the veins; this leads to œdema of the lungs and then to all the symptoms of congestive heart failure. In compensated heart disease, however, such congestive failure does not occur on attempted effort and this is due to the fact that pulmonary ventilation is reduced to about the same corresponding degree as the capacity of the heart. Peabody found that the vital capacity of patients with heart disease was reduced to 30% to 90% of the normal. Thus, there is a protective mechanism in compensated heart disease by which pulmonary ventilation fails to provide a satisfactory hydrogen ion blood content before the heart fails and induces congestive heart failure. This unsatisfactory hydrogen ion content of the blood causes general exhaustion rather than heart failure on exertion, but eventually this protective mechanism is overcome and heart failure supervenes on attempted effort or later even at rest.

### Pulse Rate After Exercise.

J. M. H. CAMPBELL (*Guy's Hospital Reports*, April, 1927) discusses the pulse rate after exercise in health and in heart disease. In health it was found that after the subject had stepped on and off a wooden block 32·5 centimetres (thirteen inches) high six, twelve, eighteen, twenty-four and thirty times per minute, the pulse rate increased proportionately to the exercise, but with wide variations. Thus after thirty steps per minute the pulse rate increase varied between 17 and 44 beats per minute in the first minute and between ten and 26 beats per minute in the second minute after exercise, that is a pulse of 72 might increase to 89 to 116 one minute after and be still at 82 to 96 two minutes after. Men and women differed little, but those in training showed a smaller increase and a quicker return to normal. In heart disease (rheumatic valvular disease) the response was sometimes normal and sometimes abnormal; it was not constant, so that it was not possible to decide by this test whether the heart was diseased or not. In effort syndrome the rise in pulse rate after exercise was greater and more prolonged than the normal and also greater than in many cases of heart disease; so that this test is of no use in distinguishing between organic heart and effort syndrome. Patients with pulmonary tuberculosis, bronchitis, emphysema and other diseases were tested. It was found that the rate at rest was high in pulmonary tuberculosis. The average was 91 compared with an average of 80 in the other patients, but the increase after exercise was as a rule no greater in any of these patients than in normal controls. Individual exceptions, of course, occurred. This exercise test was found useful only for comparing results in the same individual or for determining how much work a patient with heart disease should be allowed to do.

### Blood Pressure in Pulmonary Tuberculosis.

P. A. AND J. J. CASTILLO (*Vida Nueva*, June 15, 1927) record a series of original observations on variations in arterial and venous blood pressure in pulmonary tuberculosis. They studied arterial pressure in one hundred patients whose condition had been diagnosed as pulmonary tuberculosis by clinical, bacteriological and radiological tests. As a norm they quote statistics of observations on healthy young persons between the ages of twenty and thirty years. Hypotension is the rule in pulmonary tuberculosis. Early in the disease it is the systolic tension that is affected, later on the most important variation is in the diastolic pressure. Hence they consider variation in the systolic pressure to be of greatest importance from a diagnostic point of view and that of the diastolic for prognosis. They consider a continued diastolic pressure of below fifty millimetres as an indication of an impending fatal termination. A normal pressure or one which tends to rise is a good omen. A low or falling pressure is of serious import. An attack of pleurisy with effusion is found to cause a fall in arterial pressure. Hæmoptysis during the actual attack may be accompanied by either a high or low tension pulse. They find that every case of hæmoptysis is preceded by a rise of arterial pressure and followed by a fall. They studied venous pressure in twenty-five cases, being careful to select only patients who had no other circulatory disturbances which might invalidate the experiment. The variations noted in this series bore only slight relations to the clinical peculiarities of the conditions and varied from the findings of other investigators. They consider that logically estimation of the venous pressure should be a valuable method of investigation, but in view of the paucity of data so far available, will pass no further or more definite opinion about it.

### The Primary Infection in Tuberculosis.

PHELÉBON (*La Presse Médicale*, September 17, 1927) gives the incidence of tuberculous infection, as estimated by von Pirquet's cutaneous reaction among over three thousand inhabitants of all ages in a rural community in Normandy. The subjects for the test were carefully selected, between infancy and twenty-five years of age and were restricted to those who had lived continuously in the country and who were apparently free from tuberculous infection. The author states that it is accepted that in large cities throughout the world approximately 90% of inhabitants have been infected by the age of fifteen years and from 31% to 66% in country villages by the same age, as shown by von Pirquet's test. Among those treated by him 7·29% (123 out of 1,706 persons) gave a reaction to the von Pirquet test up to the age of eighteen years. After this age the percentage rose rapidly to 53% at twenty-one years and 61·65% at twenty-five years of age. Most of the children giving a reaction were proved to be contacts of sufferers from tuberculosis, but on the other hand many children living with tuberculous parents did not react to the von Pirquet test, though probably systematically inoculated from this source. This immunity the author attributes to the fresh air and to the powerful bactericidal action of the sun's rays, unobscured by city smoke. As a result of seven years' observation, he concludes that among true country dwellers the primary tuberculous infection occurs in the young adult rather than in the infant, proving thus an exception to the generally accepted rule. He also holds that tuberculosis commencing later among these young adults runs a milder course.

### Sanatorium Treatment of Tuberculosis.

ERNEST B. EMERSON (*Boston Medical and Surgical Journal*, September 15, 1927) discusses the treatment of pulmonary tuberculosis from the point of view of the sanatorium physician. He lays stress on the need for early diagnosis and early treatment and complains that many sanatorium patients whose condition is classified as advanced, have disregarded earlier medical advice, while the condition of others has apparently not been diagnosed correctly at a time when their disease might have been arrested. The real value of a sanatorium in early cases, he maintains, is in its education aspect. The patient should be taught the principles of hygienic living, to avoid excesses of all kinds and to regulate his life to the strictest routine on his return home. It is only in a small minority of intelligent patients under the care of a specialist, that home treatment is preferable. In the majority sanatorium treatment is indicated as a first procedure rather than as a last resort; only thus can be determined the degree of activity of the disease and the constitutional reaction of the patient. Sanatorium existence is to be accepted with the object in view of restored health and an intelligent interest in the ways and means of attaining this ideal should be stimulated in all patients. At Rutland State Sanatorium a course of thirty lectures on tuberculosis is given by the staff each year, the subjects including infection, subjective and objective symptoms, diet, climate, mental attitude and personal hygiene. The patients are invited to ask questions which are answered with absolute frankness. They are told, for instance, the significance of a cavity in the lung or the meaning of a hæmorrhage. This does not engender apprehension or gloom, but on the contrary, creates a spirit of cheerful cooperation, as the patients realize why a restricted life is essential for the arrest of the disease.

# Hospitals.

## HOSPITALS FOR THE INSANE IN VICTORIA.

THE annual report of the Inspector-General for the Insane, of Victoria, for the year ended December 31, 1926, has but recently been published. As pointed out on previous occasions in this journal, it is much to be regretted that such a long time is allowed to elapse before the report for a given period is published. This delay necessarily detracts to a large extent from the interest of the report.

### Statistics.

The admission rate and the resulting increase in the number of patients are the highest that have been recorded for some years. The daily average number of patients resident in State Hospitals was 5,467. The number of persons in Victoria certified as insane was 6,425. Apart from those in the State Hospital 86 were resident in private licensed houses, 128 were boarded out and 718 were on trial leave. In addition to these there are 128 military patients suffering from mental disease and at the end of the year there were 87 patients in receiving houses and 68 voluntary patients. The patients in receiving houses, the voluntary patients and the military patients are not included among the certified insane. The ratio of insane to the general population is put down as 1 in 266. We have pointed out in previous years that figures of this kind cannot be regarded as indicating the true state of affairs. Apart from the fact that all persons with mental disease are not certified as insane, the exclusion of the three classes of persons already mentioned is merely an indication of the approximate nature of the estimate. During the year 729 persons, 384 males and 345 females, were admitted for the first time to State hospitals. The number of readmissions was 112, 50 of these persons being males and 62 females. The total number of persons admitted was therefore 841. Of these 117 were classified as suffering from primary dementia, 118 from delusional insanity, 89 from congenital insanity (twelve were epileptics), 99 from senile dementia, 65 from secondary or terminal dementia, 36 from general paralysis of the insane, 37 from epilepsy and 21 from gross lesions of the brain. It is pointed out that these figures indicate that 70% of the persons admitted to State hospitals had no hope of recovery or even of discharge to authorized care.

Interesting information is gathered from a table in which are set out the probable causes of insanity in the patients admitted during the year. Forty-six of the 841 patients admitted are regarded as owing their insanity to the excessive use of alcohol and 41 to venereal disease. Old age was responsible for the insanity of 101 persons and in 107 some congenital defect was discovered. Hereditary influences were ascertained in 81 patients. The Inspector-General points out that in 11% of the admissions no known causation was present and that therefore the figures dealing with the causation are lower than they should be.

During the year 292 persons were discharged from State hospitals; 169 are recorded as having recovered, 101 as having been relieved and 22 as manifesting no improvement. No indication is given of what is meant by recovery.

### Pathological Work.

*Post mortem* examinations were made in 75% of the 403 persons who died; the most frequent immediate causes of death were senile decay, heart disease and pneumonic conditions.

A separate report is made by Dr. W. A. T. Lind, the pathologist of the Department. He mentions his investigations into manic depressive insanity and insanity with epilepsy which have been published in this journal. He refers with satisfaction to the system of routine *post mortem* examination which exists in Victoria. This system has enabled him to amass more than 3,000 records of the different types of insanity and these records are always available for reference and study. He points out that at the present time investigation of disease is possible from the point of view of morbid pathology only and he strongly advocates the creation of a research hospital in order that clinical pathology may be examined in the mass. He finds that an intensive study of any particular type of insanity cannot be undertaken in hospitals at present, because it would require the undivided attention of one investigator and the aid of a pathological assistant in order to make the work complete and valuable. He argues this question at some length and in a manner which must convince any thoughtful person. Whether it will have any effect on the stony hearts of the politicians who hold the money bags, is quite a different matter. The Inspector-General lays emphasis on the need for more accommodation on account of overcrowding, on the necessity for improved sanitation and on the desirability of providing better facilities for recreation and amusement. When these matters are neglected it is unlikely that Dr. Lind will get his special hospital. If more accommodation is to be provided, there is no reason why it should not be provided in a special way. Research in mental disease will bring improved methods of treatment; improved methods of treatment mean shorter duration of a patient's stay in hospital at any rate in some forms of insanity; a shortened stay means a saving in money. If the politicians could be made to realize this, they might open the money bags.

# University Intelligence.

## THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on February 6, 1928.

The following degrees were conferred *in absentia*:

*Doctor of Medicine* (M.D.): Warnford Moppett.

*Doctor of Dental Science* (D.D.S.): Edward William Haggett.

Dr. Moppett graduated M.B., Ch.M., in 1923 and the degree of M.D. was awarded him for his thesis entitled: "An Investigation of the Reactions of Animal Tissues Exposed to Monochromatic X Rays of Different Wave Lengths."

Dr. Haggett graduated Bachelor of Dental Surgery in 1921 with first class honours and the University Medal. He was awarded the doctorate degree for his thesis entitled: "The Effect of Pulpless Teeth on Immunity and its Relationship to Public Health and Preventive Medicine." He is the first graduate of the Faculty to obtain this degree.

The Chancellor, on behalf of the Senate, extended a hearty welcome to the new Vice-Chancellor, Professor R. S. Wallace.

The Senate decided to place on record its cordial congratulations to the Deputy Chancellor, the Honourable Sir Philip Street, K.C.M.G., Chief Justice of New South Wales, on the high honour recently conferred upon him by His Majesty the King.

In response to an invitation from the Royal College of Physicians, the Senate decided to appoint Dr. C. Bickerton Blackburn as delegate of the University on the occasion of the tercentenary of the publication of William Harvey's treatise, "De Motu Cordis," to be held in London.

Provisional approval was given for the inauguration of the University War Memorial Carillon on Anzac Day, April 25.

The following examiners were appointed for the forthcoming Final Degree Examination in Medicine: *Medicine*, Professor A. E. Mills, Dr. Cecil Purser; *Surgery*, Professor F. P. Sandes, Dr. H. S. Stacy; *Obstetrics*, Professor J. C. Windeyer, Dr. S. H. MacCulloch; *Gynæcology*, Dr. R. Davies, Dr. G. Armstrong; *Clinical Medicine*, Dr. Sinclair Gillies, Dr. A. W. Holmes à Court; *Clinical Surgery*, Dr. J. L. McKelvey, Dr. C. E. Corlette.

# Research.

## THE FOULERTON RESEARCH PROFESSORSHIP.

THE ROYAL SOCIETY has recently advertised in our columns the appointment of the second Foulerton Research Professor and invites candidates of "sufficient distinction" to apply for the office. The following are the regulations.

1. Appointments will be made by the President and Council of the Royal Society on the recommendation of a specially appointed committee of Fellows called "The Foulerton Research Fund Management Committee."

2. The duties of the holder of a Foulerton Research Professorship will be to conduct in a place approved by the Committee such original researches in medicine or the contributory sciences on lines approved of by the Committee as shall be calculated to promote the discovery of the causes of disease and the relief of human suffering.

3. In awarding professorships regard shall be had primarily to the ability of the individual rather than to the subject of his research, provided only that the subject of research is one permitted by the regulations. Professorships shall be awarded only to candidates who have shown outstanding ability for independent research.

4. It shall be the duty of a professor to devote his whole time to research, except that he may give a limited course of instruction in the subject of his research to advanced students and any award shall be made subject to its being found possible to make suitable arrangements for the research being carried out at a university or other approved place.

5. The stipend of a professor, except as provided in Regulation 6 below and except for the contribution from his stipend required under the scheme mentioned in Regulation 11 below, shall be at least £1,400 *per annum*.

6. In special cases, Regulations 4 and 5 notwithstanding, the President and Council may appoint to a professorship the holder of a paid academic or other scientific appointment, provided they are satisfied that the duties of such other appointment occupy only a subsidiary portion of the applicant's time and that its retention would not interfere with the discharge of the duties of the professorship as essentially a whole-time appointment. In such cases they may order the payment of such stipend as they may think fit, provided that the total annual income of a professor from such paid appointment, together with the stipend paid him from the Foulerton Research Fund shall not be less than £1,400, except for the contribution from his stipend required in Regulation 11 below. No Royal Society Professor shall accept or hold any appointment, paid or otherwise, other than his Royal Society Professorship, except with the knowledge and approval of the President and Council.

7. A professor shall be appointed for a period of five years, the appointment being renewable on the application of the professor for further successive periods of five years, provided that the President and Council are satisfied that the professor is competent to discharge the duties of his professorship and is not neglecting these duties, except that when a professor is over fifty-five years of age, appointment or renewal shall be only for the period until he attains the age of sixty years.

8. Save as is hereinafter in this Regulation provided a professor shall vacate his professorship on attaining the age of sixty, except that the President and Council shall have power, by the vote of two-thirds of the members present at a meeting of the Council held within six months before the date on which the professorship would otherwise be vacated, to extend the appointment for a period not exceeding three years and so on from time to time.

9. In special cases, Regulations 7 and 8 notwithstanding, a professor may be appointed for a limited period of years without the opportunity of renewal at the end of this period, but the level of ability demanded in these special cases must not be lower than that demanded from other Royal Society professors.

10. If at any time in the opinion of the President and Council a professor is found to neglect or to be unable to discharge the duties of his appointment or to be guilty of conduct or of continuing a course of conduct unbecoming in the holder of a Royal Society professorship, the President and Council shall have power at once to terminate his professorship.

11. Unless the President and Council order to the contrary, provision shall be made for the superannuation of each professor under a contributory scheme to be approved by the President and Council.

12. Should a member of Council or of the Management Committee become a candidate for a professorship, he shall *ipso facto* vacate his membership of Council or of the Management Committee. A professor shall not be eligible for service on Council or on the Management Committee during his tenure of a professorship.

13. The President and Council may make grants to a professor towards the various expenses incurred in his research, such as the purchase of apparatus and payment for assistance, provided they are satisfied that it is not possible to meet these expenses from other sources.

14. Members of all nationalities and of both sexes shall be eligible and in these regulations words importing the masculine gender shall include the feminine gender.

15. The President and Council reserve the power from time to time to make new regulations for the administration of the Foulerton Research Fund.

16. Candidates should state their age and the lines of research in which it is proposed to engage.

17. Applications (marked outside "Foulerton Professorship") must reach the Royal Society not later than September 30 next.

## DEATHS UNDER LOCAL ANÆSTHESIA.

PROFESSOR G. CANUYT, of the University of Strasburg, is endeavouring to collect information concerning deaths caused by local anæsthetics. He asks medical practitioners and particularly those engaged in ear, nose and throat work, who have witnessed deaths during the course of local anæsthesia or who have knowledge of such accidents to communicate to him certain details. He undertakes not to divulge the names of those who are kind enough to write to him on the subject. The information sought is as follows:

(i) The sex, age and other general characters of the patient.

(ii) The diagnosis of the affection on account of which the patient was under treatment.

(iii) The nature of the operation for which the local anæsthesia was induced.

(iv) Whether death occurred before, during or after the surgical procedure.

(v) The conditions under which the accident occurred:
- (*a*) The anæsthetic used;
- (*b*) The dose and concentration;
- (*c*) The method of application (injection, painting on the surface).

(vi) The symptoms supposed to have been caused by the anæsthetic.

Professor Georges Canuyt wishes to thank those practitioners who will accede to his request. The replies should be sent to him at the "Clinique Oto-rhino-laryngologique, Hôpital Civil, Strasbourg, Alsace."

# Obituary.

## DAVID JOHN STEWART BURT.

WE regret to announce the death of Dr. David John Stewart Burt which occurred at Sydney on February 8, 1928.

## ROBERT OLIVER DOUGLAS.

WE regret to announce the death of Dr. Robert Oliver Douglas which occurred as a result of a motor car accident at Hamilton, Victoria, on February 9, 1928.

## Proceedings of the Australian Medical Boards.

### NEW SOUTH WALES.

THE undermentioned have been registered under the provisions of *The Medical Act* 1912 *and* 1915 of New South Wales, as duly qualified medical practitioners:

Sheehan, William Beverley James, M.B., B.S., 1927 (Univ. Sydney), England.

Susman, Maurice Philip, F.R.C.S. (England), 1925, 215, Macquarie Street, Sydney.

Change of name:
Felstead, Muriel, to Barnes, Muriel (Byron Bay).

## Books Received.

INTERNATIONAL HYGIENE, by C. W. Hutt, M.A., M.D., D.P.H.; 1927. London: Methuen and Company, Limited. Royal 8vo., pp. 261. Price: 10s. 6d. net.

A TEXT-BOOK OF PRACTICAL THERAPEUTICS, by Hobart Amory Hare, B.Sc., M.D., LL.D.; Twentieth Edition, enlarged, thoroughly revised and largely rewritten; 1927. Philadelphia: Lea and Febiger. Royal 8vo., pp. 1094, with illustrations. Price: $7.50 net.

MAX VON PETTENKOFER: HIS THEORY OF THE ETIOLOGY OF CHOLERA, TYPHOID FEVER AND OTHER INTESTINAL DISEASES: A REVIEW OF HIS ARGUMENTS AND EVIDENCE, by Edgar Erskine Hume, M.D., Dr.P.H., LL.D.; 1927. New York: Paul B. Hoeber Incorporated. Post 8vo., pp. 156, with illustrations. Price: $1.50 net.

CONVALESCENCE, HISTORICAL AND PRACTICAL, by John Bryant, M.D.; 1927. New York: The Sturgis Fund of the Burke Foundation; Royal 8vo., pp. 285, with illustrations.

OUTLINES OF DENTAL SCIENCE, Volumes VII and VIII; Dental Surgery, by J. L. Dudley Buxton; 1927. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 194 and 173, with illustrations. Price: 7s. 6d. net each.

## Diary for the Month.

FEB. 20.—New South Wales Branch, B.M.A.: Organization and Science Committee.
FEB. 21.—Tasmanian Branch, B.M.A.: Council.
FEB. 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEB. 22.—Victorian Branch, B.M.A.: Council.
FEB. 23.—South Australian Branch, B.M.A.: Branch.
FEB. 24.—Queensland Branch, B.M.A.: Council.
FEB. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAR. 1.—South Australian Branch, B.M.A.: Council.
MAR. 2.—Queensland Branch, B.M.A.: Branch.
MAR. 6.—Tasmanian Branch, B.M.A.: Council.

## Medical Appointments.

Dr. John Grieve Woods (B.M.A.) has been appointed Government Medical Officer at Urana, New South Wales.

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Dr. Ernest Leslie Morgan (B.M.A.) has been appointed Principal Microbiologist, Office of the Director-General of Public Health, Sydney.

## Medical Appointments Vacant, etc.

**FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx.**

COMMONWEALTH OF AUSTRALIA: Assistant Medical Officer, Nauru.

ADELAIDE HOSPITAL: Medical Registrar, Surgical Registrar.

## Medical Appointments: Important Notice.

**MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.**

| BRANCH. | APPOINTMENTS. |
|---|---|
| NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney. | Australian Natives' Association.<br>Ashfield and District Friendly Societies' Dispensary.<br>Balmain United Friendly Societies' Dispensary.<br>Friendly Society Lodges at Casino.<br>Leichhardt and Petersham Dispensary.<br>Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney.<br>Marrickville United Friendly Societies' Dispensary.<br>North Sydney United Friendly Societies.<br>People's Prudential Benefit Society.<br>Phœnix Mutual Provident Society. |
| VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne. | All Institutes or Medical Dispensaries.<br>Australian Prudential Association Proprietary, Limited.<br>Mutual National Provident Club.<br>National Provident Association.<br>Hospital or other appointments outside Victoria. |
| QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane. | Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing.<br>Brisbane United Friendly Society Institute.<br>Stannary Hills Hospital. |
| SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide. | All Contract Practice Appointments in South Australia.<br>Booleroo Centre Medical Club. |
| WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth. | All Contract Practice Appointments in Western Australia. |
| NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington. | Friendly Society Lodges, Wellington, New Zealand. |

**Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.**

## Editorial Notices.

**MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.**

**All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)**

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